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IN MEMORIAM

DOCTOR STANFORD E. CHAILLÉ

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NEW ORLEANS

Once again we are assembled fittingly to make manifest our enduring reverence for the memory of one of the greatest men of medicine of our time, Stanford Emerson Chaillé—the mere mention of whose name evokes in all of us the tenderest of emotions and impels us pause to contemplate in admiration and with deep respect a life so gloriously well spent, while out of the depths of our memories, well-up pleasing reflections of those many happy moments in our own lives, made blessed by their contacts with his.

That our distinguished President, Dr. Edwin L. Zander, should assign me to speak for a few moments here upon Dr. Chaillé's life and my personal relations with him as a small contribution to this memorial, is to have conferred upon me an honor far beyond my meager ability, or my merit; and it is therefore with temerity that I approach the task. However, since it is, I am sure, in no sense expected of me to add by my poor oratory even a scintilla to the lustre of Dr. Chaillé's fame, I do believe I may be privileged at least to epitomize a few of the high-lights in his marvelous career, for the benefit of those of us who have but recently become affiliated with our Society and in consequence are little acquainted with his life, filled as it was with so many magnificent achievements, as a result of which his country, his fellowmen and medicine are so justly proud of him.

After graduating from the medical department of the University of Louisiana, now Tulane, his studies took him abroad, where in Paris he studied physiology under the great Claude Bernard. In 1859 he was demonstrator of anatomy here. In the early days of the War Between the States, he was acting Surgeon General of Louisiana after which he was the Medical Inspector of General Bragg's army in Tennessee, then later he was in charge of a hospital in Atlanta, from which place he was ordered to Macon, there again heading a hospital. He was taken prisoner at Macon, and after the cessation of hostilities resumed his activities in this city. A paper of his on Medical Jurisprudence, written by request, when read at the International Medical Congress in Philadelphia, immediately prompted overwhelming and well merited praise from both the President and Congress of the United States. He served as Secretary of a board of twelve appointed by Congress to investigate yellow fever and its cause. He was chairman of the group of four constituting the Havana Yellow Fever Commission appointed by the National Board of Health, later becoming a member of the National Board of Health appointed by President Arthur, being also its Inspector General in New Orleans. He was the moving spirit behind the enactment of Medical Laws in Louisiana. He was one of the founders and charter members of this Society. He succeeded Dr. Richardson as Dean of the Medical Department of Tulane University, in which capacity he served for almost a

*Nineteenth Annual Stanford E. Chaillé Oration, delivered on November 6, 1944, before the Orleans Parish Medical Society.

quarter of a century, passing to his eternal reward, May 27, 1911. It was not his mere participation in these events just recounted that made Dr. Chaillé the great man that he was, but rather his unremitting zeal, deep comprehension of purpose and his indefatigable devotion to the duties involved, toward each of which his great energies were bent with firmness and the full conviction that in the end his every high hope and noble resolve would merit the crown of success.

During my attendance at Tulane Medical School he was Dean and also filled the Chairs of Pathology, Physiology and Hygiene. My personal relations with Dr. Chaillé were always most agreeable and pleasant, and numerous enough, even at this late hour, to permit me, without the exercise of my imagination to recall quite vividly his colorful and commanding personality, as well as the impressions that he made upon me and I might also say upon my fellow students. In the early periods of my attendance at his classes all of my fellow students and I looked upon him in great awe, not because he was in any way austere or distant in his manner towards us, but rather because we seemed to sense in him a living encyclopedia of all medical lore. But we soon began to recognize his masterful knowledge of the subject matter of his lectures, his unequalled pedagogic technics and his profound psychologic procedures, which at all times intrigued our interest and held our undivided attention, while we sat spellbound in his presence.

And over and above all these, he manifested toward all of us a parental interest that was well calculated to inspire each of us to greater effort; indeed his influence upon us was most wholesome.

I am sure that each of my fellow students and I will today acknowledge with profound gratitude the profit that must of necessity have accrued to each of us, as a result of the application in our daily lives of the sublime principles which, as our Dean, Dr. Chaillé so ardently endeavored to inculcate in his various dictums, through many of which by subtle suggestion he sought to arouse our impulses to highest ideals and kindle our intellects to greater endeavors. I can recall with what indescribable urge he exhorted us to the exercise of our own initiative, that great force through which alone all the hidden truths of medicine have been unearthed.

You will pardon, I feel sure, my allusion to the pride that seemed to suffuse our very being, when he with unerring accuracy pictured to us the unique and sacred relationship that a physician bears to his patient, at which moment his very spirit seemed alight with the hope that we would never violate it.

May I also add how shortly before our graduation our young and eager hearts throbbed with emotion when he in most solemn tones invoked that ancient oath of Hippocrates, every provision of which he then assured us was as applicable in our lives as it had been in the lives of those among whom it was first ordained and uttered.

I recall his abhorrence to all ironclad dogmas, cults and isms in medicine, his maintenance of the right of another, no matter how humble or obscure he may seem, to hold such view as in his opinion, earnestly held, seemed correct and true, and his firm advocacy of the right of medical men to organize and his ardent love for our beloved profession.

Friends, I could go on for hours recalling to memory the many happy occasions that graced my life in his presence, and proclaiming the virtues, intellectual abilities and mental traits of Dr. Chaillé, who, as all of us must now know, many times by his presence and words of wisdom lent grace and dignity to many of our gatherings, but time does not permit. In no more fitting manner, I am sure, may we dignify this occasion than by the perpetuation of this our yearly custom of listening to the voice of our beloved science, as she eloquently speaks with authority through the lips of one of her illustrious sons, one of whom, the distinguished Dr. Kolmer of Temple University, will honor with us the memory of Dr. Chaillé by his presence here tonight.

In closing may I hold up to the view of those of us who are young in this our field of hope, labor and service the life of Dr. Chaillé as one they should endeavor to emulate, remember

"The lives of great men all remind us
We too can make our lives sublime
And departing leave behind us footsteps on the sands of time."

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ATYPICAL AMEBIC COLITIS*

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and
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In any presentation of atypical forms of a common ailment, the natural expectation would be to hear about unusual or bizarre manifestations of the particular disease. When amebiasis is the subject of the discussion, this expectation is not warranted. Since amebiasis is as great a mimic of abdominal disease as syphilis is of systemic disease, and atypical forms of one sort or another perhaps outnumber the so-called typical cases, the common deviations from the classical picture, as well as more unusual forms should be included under this title.

What is meant by a "typical" case of amebic colitis? Such a case would probably conform to the description given in any standard textbook of medicine. Unfortunately for diagnosis, this description frequently does not hold good. From the public health standpoint, on the other hand, this discrepancy is perhaps an advantage, since it has been estimated that the protozoon is harbored by about 10 per cent of the general population, a large segment to be disabled by a single disease, if all had the text-book form.

There is great variability of leading symptoms. Although diarrhea is accepted as the standard symptom, close to 50 per cent of patients with proved amebiasis give a history in which constipation is featured. Some cases of long duration, say 10-20 years, tell of diarrhea at the onset, followed by intermittent diarrhea, but a generally constipated condition. Mild abdominal distress with or without a history of an occasional loose stool is a common symptom. One rather recent patient, a 21 year old girl had a chief complaint of dysmenorrhea, with no symptom referable to the bowel. The gynecologic approach to her problem was fruitless, but anti-amebic therapy brought marked amelioration. Clinically, amebic colitis commonly simulates chronic cholecystitis, cholelithiasis, peptic ulcer, and pancreatitis. The full syndrome of intestinal obstruction can be produced by the granulomatous form of the disease, and laparotomy after a preoperative diagnosis of colonic neoplasm has been a not uncommon error—error, since emetine therapy results in a dramatically rapid lysis of the granuloma with consequent relief of the obstruction.

Amebic colitis can be a hyper-acute disease of extreme gravity. Three cases of this so-called ulcero-necrotic form were recently described by the authors.[1] The entire life span of the patient, from the onset of symptoms to death may be less than a week. A rapidly fatal exacerbation may occur in a long standing case. These cases are to be differentiated from more conventional forms in which severe complications may eventuate, but are fulminating from the onset and unaffected by specific therapy. This type of case is rather rare in this country, but has been described as occurring in China and the Philippines by Faust,[2] who suggested that outlanders becoming infected in locales of hyperendemicity are the individuals liable to contract this serious form of the disease.

Included in the aforementioned 10 per cent are many individuals unaware of their harboring the ameba. These are customarily referred to as carriers, but the accuracy of this designation is open to question, since they may at any time develop some clinical manifestation of the disease. Some patients have no complaint more specific than lassitude, frequently of years' duration, not knowing the experience of

*Read before the Orleans Parish Medical Society, October 9, 1944.

good health, which comes as a revelation after successful anti-amebic therapy.

With increasing experience it is becoming more and more apparent that there is no truly typical case of amebiasis, that the disease is protean, widespread, and must ever be borne in mind as a diagnostic possibility.

There is a parallelism between the variability of clinical picture and the variability of pathology, which may or may not be reflected by the proctosigmoidoscopic appearance of the lower bowel. What is the common conception of the pathology of amebic colitis? Quite correctly one brings to mind a process in which there are discrete, undermined, flask-shaped ulcerations with normal or nearly normal intervening mucous membrane. The ulcer base overlies submucosa, muscularis, or serosa, depending on the depth of penetration. Cysts and trophozoites of *Endameba histolytica* may be seen in the necrotic centers and in the surrounding tissues. The cecum and rectosigmoid are the areas of predilection of the lesions. This might be considered the typical pathology, which may be complicated relatively rarely by hepatitis and abscess of the liver or perforation and peritonitis. Actually great variation from this picture is commonplace. There may be no more than a few areas of superficial ulceration in the entire colon. At the other extreme there is the ulcero-necrotic type of amebiasis, in which there is deep, diffuse, confluent, serpiginous ulceration, producing a condition of the bowel difficult to distinguish proctoscopically from chronic ulcerative colitis in aggravated form. In these cases sloughing of whole sections of the bowel may be encountered. In one of these cases reported by the authors the entire posterior wall of the transverse colon sloughed out, and at autopsy it was seen that the posterior bowel wall was made up of matted loops of small intestine. In another of these cases a piece of tissue identified histologically as nondescript necrotic material was passed by rectum. Figure 1 shows the deep undermining penetration of a necrotizing amebic ulcer, illustrating the process by which sloughing takes place. It is interesting and perhaps important to note here that two of the three cases of ulcero-necrotic amebic colitis reported had a complicating infection due to *B. dysenteriae*, one a Flexner and the other a Shiga. The question arose as to which pathogenic organism played the dominant role, whether the amebae caused necrosis by blocking the intestinal blood supply, or whether the necrosis was caused by the bacterial toxin, or whether there was an additive or synergistic effect.

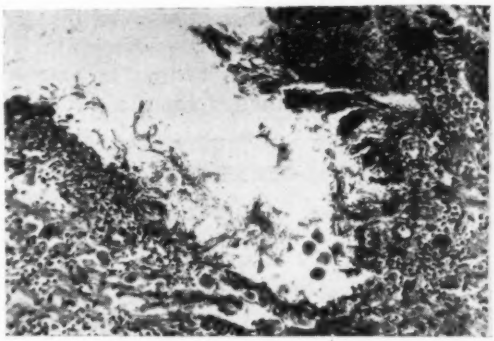

Fig. 1. Section through ulcer of colon showing penetrating necrosis and numerous trophozoites of Endameba histolytica throughout the submucosa and muscularis. Lateral dissemination predisposes to sloughing of superficial layers.

Another unusual but distinctive form of amebic infestation of the bowel is the type characterized by tumor formation. In some individuals the infestation provokes a response characterized by the production of granulation tissue. Depending on the rate of growth of the tumor, acute or chronic intestinal obstruction is observed. The roentgenographic appearance is indistinguishable from carcinoma. Because of the affinity of the protozoon for the cecal and rectosigmoidal regions, these are the predominant sites of amebic granulomata. There are many reports of cases of amebic granuloma explored in quest of a neoplasm. Nino[3] reported a case of a patient so diagnosed preoperatively who, following resection of the cecum and ascending colon, died of peritonitis. At autopsy the pathology was established as "ulcero-necrotic intestinal amebiasis with amebic tumor of the cecum and ascending colon." The authors

observed amebic granulomata in a husband and wife. The wife underwent a resection of the cecum for what purported to be a neoplasm (fig. 2) the true nature of the disease being revealed only after pathologic examination of the tissue. The husband

Fig. 2. Barium enema demonstrating cecal filling defect.

more fortunately presented his granuloma within reach of the sigmoidoscope, and the diagnosis was made by biopsy. Histologic study of tumors of amebic etiology reveals the presence of the organism, usually in great numbers throughout the granuloma.

It might be charged that this has been no more than a brief broad discussion of amebiasis, without emphasis on the atypical. If atypical is made synonymous with unusual, the authors must plead guilty. If, however, it has been demonstrated or only suggested that the atypical is truly the usual in amebiasis, that amebiasis is a disease of protean nature, and a diagnostic pitfall, the purpose of this presentation will have been achieved. The fact to be emphasized is that because of its manifold symptomatology, amebiasis should always be considered in differential diagnosis.

REFERENCES

1. Silverman, D. N., and Leslie, A.: Intractable amebic colitis, with special reference to the ulcero-necrotic forms, J. A. M. A. In Press.

2. Faust, E. C.: Some modern concepts of amebiasis, Trans. and Studies of College of Physicians of Philadelphia, 4 Ser., Vol. II, No. 3, December 1943.

3. Nino, F. L.: Amebiasis intestinal ulcero-necrotica y tumor del ciego y colon ascendente, Novena reunion, Soc. argent. de pat. reg: 2:813, 1937.

DISCUSSION

Dr. E. Carroll Faust: The surface of a primary lesion shows a round, somewhat reddened elevation with a pinpoint center. This is the actual site of entry for the ameba as it digests its way into the intestinal wall. If one wishes to visualize where the amebic lesions are most frequent in an average individual, he will discover that they preponderate at the levels of primary location. However, in the large bowel there is always the opportunity for the progeny from the original colonies to establish secondary lesions. The cysts which are found in formed stools cannot penetrate the tissues until they have excysted and the stage found in the tissues is always the active vegetative one.

A diagrammatic representation of the number of lesions in an average individual at different levels of the bowel indicates that in the cecal area (that is, the cecum, appendix and adjacent portion of the ascending colon), there is on the whole the largest number of lesions, while in the sigmoidorectal area, which can be visualized with the proctoscope, there is the second largest number. There are usually relatively few lesions in the intermediate parts of the colon, even at the flexures, compared with the heavily infected site at both ends of the large bowel. Undoubtedly the largest number of individuals have primary lesions in the cecal area, but later, as the progeny pass down and produce colonies at lower levels, the colon and rectum become increasingly involved. This explains why such a large number of these chronic cases of amebic colitis have ulcerating lesions, cicatrices and amebomas in the proctodeal level which can be visualized by sigmoidoscopy.

Dr. Gordon McHardy (New Orleans): After listening to the excellent presentation by Dr. Silverman and Dr. Leslie and the instructive discussion by Dr. Faust, it seems almost impossible for anyone to add further to the subject. However, there is one point I feel Dr. Faust dislikes which came into interest recently with us and I would like, if possible both Drs. Silverman and Leslie and Dr. Faust to add discussion on this one atypical point we have found occurs in amebiasis. It is commonly believed that fever is not a part of uncomplicated amebiasis; however in 790 cases reviewed last year 52 cases, uncomplicated as far as it was possible to determine, showed fever. Of these 52 cases there were acute amebic dysentery manifestation in twelve; chronic dysenteric symp-

toms in 32, and so-called carrier state in the remainder. I think the febrile issue is of interest when one considers atypical amebiasis. The fact that fever has a relation to amebiasis, is demonstrated by the fact that these patients become afebrile after satisfactory therapy.

The essayists have presented an excellent paper which brings attention to the fact that amebiasis has serious complications which everyone should consider. If right in the assumption, I think only six cases in our series were similar to those presented tonight. Two of these, I know were Dr. Silverman's cases. Of the six, two were operated on and both recovered. Certainly it would have been disastrous if either had died, after having had a major abdominal procedure, from an illness correctable by medical measures. With an incidence of amebiasis in 16 per cent of examined patients in our study, the overwhelming importance of an abdominal search for amebiasis in all abdominal cases is imperative.

Dr. D. C. Browne (New Orleans): Dr. Silverman and Dr. Leslie have called attention to a clinical occurrence we will probably see more frequently. An infection involving 10 to 16 per cent of our population is entitled to more consideration than now given by the thinking physician. Certainly, an infection capable of producing these complications, mortality and morbidity, should be treated more thoroughly and carefully, for it is not at all improbable that such amebic granulomas as presented here are the result of inadequate treatment. I am convinced that the ease with which symptoms may be relieved with the present excellent therapy has promoted negligence.

It must be borne in mind that only a small percentage of gastrointestinal symptoms the amebiasis case presents are the direct result of the infection; less than 39 per cent may be related and the findings of *Endameba histolytica* in no sense relieves the clinician of his responsibility in making a careful gastrointestinal study. Evaluation necessitates careful clinical judgment.

Dr. J. A. Colclough (New Orleans): It is elementary to say one can have amebic abscess without amebic dysentery. I quite agree with the essayists that amebiasis is the great imitator of the abdomen. It was my good fortune to read, during the past few months, something of the use of thorotrast in the diagnosis of liver conditions. I removed a gallbladder in a young woman in March, which was full of stones. She made an uneventful recovery and became febrile four weeks after operation. Temperature was septic and an internist and I disagreed between the presence of subphrenic abscess and liver abscess. Repeated examinations of stools were negative for amebae. We injected thorotrast intravenously. Two hours later a picture of the liver and spleen showed, with diagrammatic clarity, a circular area of lessened density of the liver. Aspiration revealed presence of liver abscesses, which had been suspected and could not be proved. I would like to recommend use of thorotrast in diagnosis of atypical amebiasis and obscure abdominal conditions.

Dr. D. N. Silverman (In closing): I wish to thank Drs. Faust, McHardy, Browne and Colclough for their discussion of this paper. I believe that our demonstration provides clinical and pathologic corroboration of Dr. Faust's outstanding experimental work on the sites of amebic invasion of the colon. The most acute case of ulcero-necrosis of the colon in our experience was a massive slough of the entire transverse colon. The man died two days after onset. This man was also infected with the dysentery bacillus. That brings up the question whether or not many of these fulminating cases with severe symptomatology and early death are complicated by previous or concomitant infection of the individual by the dysentery bacillus.

In answer to Dr. McHardy: In a survey of a large series of cases there must be considered the complications of chronic amebic colitis. We know now that hepatitis is quite frequent without localized abscess formation and can be the cause of fever. I do agree that some of our own patients have had fever, even before a complication was demonstrated.

Dr. Browne mentioned adequate therapy of amebiasis. I am disheartened over the attempts at adequate therapy as employed today, since many of our cases of chronic, long-standing amebic colitis refuse to respond to all the known drugs now used in anti-amebic therapy.

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THE DIAGNOSIS AND TREATMENT OF ACUTE DIARRHEAL DISORDERS*

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Physicians in the active practice of medicine have given little attention to the etiologic diagnosis of the acute diarrheal disorders. This group of diseases can not be diagnosed with accuracy clinically, and the necessary laboratory procedures were in the past complicated and to a great extent unsatisfactory. A further deterrent was the fact that specific therapeutic measures were not available, and thus there was no practical importance to the diagnosis of a

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single case on etiologic rather than symptomatic grounds. Recent progress in laboratory technic has now given us a simple but effective diagnostic test, and studies on the effectiveness of sulfonamide preparations in various intestinal infections have clearly shown that an etiologic diagnosis is essential if the patient is to receive the best type of medical care.

ETIOLOGY

To begin with, it has been found that the great majority of all acute diarrheal disorders which come to the attention of the physician in this country are infectious in nature and are caused by a member of either the *Shigella* or the *Salmonella* group of organisms.

The importance of these two groups as a cause of acute diarrhea is shown in the two charts which follow. The first (chart 1)

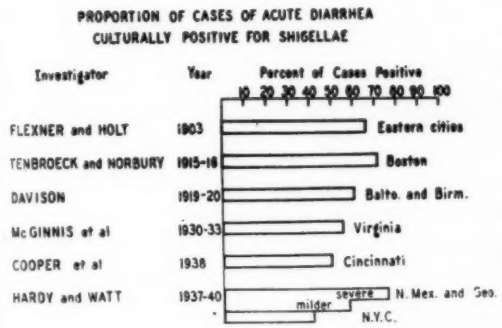

Chart I. The proportion of acute diarrhea found positive for *Shigella* by different investigators since 1900 in different areas of the United States.

gives the diagnostic results obtained by various workers in this country since 1900. The relatively slight variation in findings, in spite of different laboratory technics, is striking.

Of more interest to Louisiana physicians are the results shown in chart 2. These are selected portions of our own studies. In it are given the laboratory results of fecal cultures obtained from cases of acute diarrhea in several geographic areas. Except in the New Mexico-Georgia series these are hospital and outpatient cases, in other words, patients sufficiently ill to seek medical care on their own initiative. The uniformity of results in New Mexico, Georgia, Puerto Rico and Shreveport stand in marked contrast to those obtained in the New Orleans Charity Hospial. *Salmonella* infections, which were

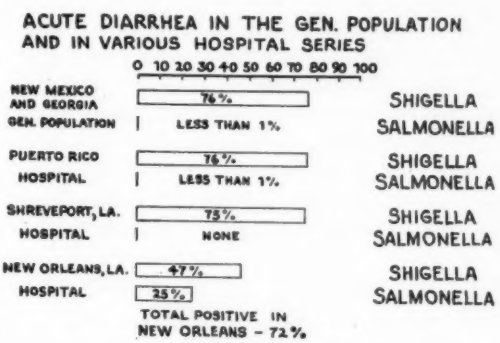

Chart II. The percentages of acute diarrheal disorders culturally positive for *Shigella* and *Salmonella* in selected general population studies and in hospitalized cases in different georgraphical areas.

an unimportant fraction of the total cases seen in most areas, make up at least 25 per cent of the cases studied in New Orleans up to this time.

SULFONAMIDE THERAPY

The practical importance of these observations lies in the therapeutic response of these different organisms to sulfonamide therapy.

The results of recent studies may be summarized as follows: For *Shigella* infections there are effective specific therapeutic measures although there are important differences in the response of different varieties to the drugs employed. Against the *Salmonella* infections we do not have such effective agents.

Sulfonamide therapy in shigellosis got its first big impetus from the development and application of sulfaguanidine in the treatment of intestinal infections. Its use has been widespread and many other preparations have also been utilized. In the study, the findings of which are partially summarized on the following charts, two criteria of effectiveness were employed; first, the clinical response of the individual to treatment, and second, the results of daily fecal cultures with colony counts of patho-

genic organisms. The latter procedure is a far more accurate index of the effectiveness of a drug. It is a truly objective measure and not subject to individual interpretation, and it is therefore the one relied on primarily at the present time.

One point a little aside from the main question of therapeutic effectiveness is of interest. There has been considerable discussion on the merits of readily absorbed versus poorly absorbed sulfonamides in the treatment of enteric tract infections. Chart 3 shows the level of dissolved sulfonamide compared with the total sulfonamide levels in feces. It is important to note that the absorbed compounds such as sulfadiazine are present in the stool and that the level of dissolved drug, the therapeutically active portion, is higher than that sought in the blood for treatment of parenteral infections. The absorbed compounds are therefore able to attack both the organisms in the lumen of the bowel but also those more intimately associated with the mucosa and glands.

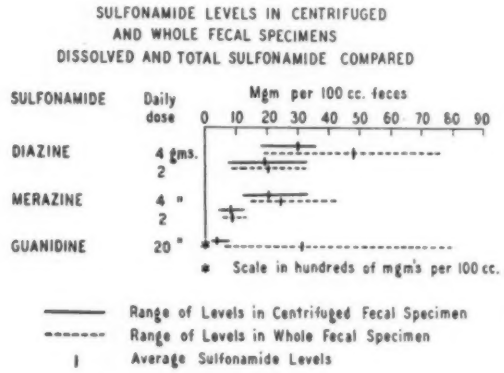

Chart III. Sulfonamide levels found in fecal specimens showing range of levels in centrifuged (dissolved therapeutically active portion) and in whole (total sulfonamide) specimens.

The observation makes much less surprising the data in chart 4, which show the therapeutic results in seven groups of cases of *Shigella paradysenteriae* Flexner infections treated with seven different sulfonamide preparations, as compared with the results in an untreated group of similar cases. All the sulfonamides tested were of definite value in eliminating the organisms from the intestinal tract. The most effective among those tested was sulfadiazine and least effective was sulfaguanidine. In this particular group all individuals treated with sulfadiazine had negative stool cultures by the end of the third day of treatment; 95 per cent of the controls were still positive at the end of the same period of time. The other preparations used, sulfapyrazine, sulfamerazine, sulfamethazine, sulfathiazole and sulfasuxidine, were in general effective in the order named with little variation between them.

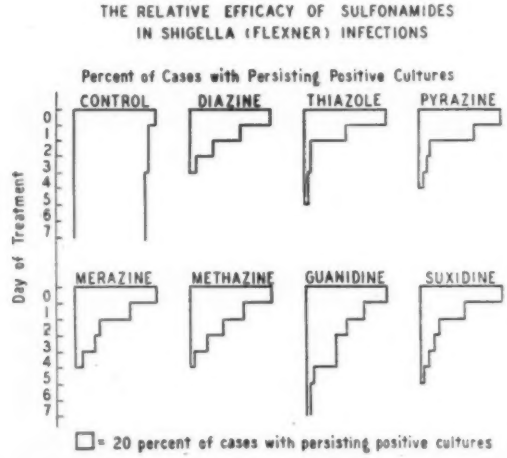

Chart IV. The efficacy of seven different sulfonamide preparations in the treatment of *Shigella paradysenteriae* Flexner infections as indicated by the percentage of continuing positive cultures.

In chart 5 is compared the response to sulfadiazine in epidemics due to three different members of the *Shigella* group. The curves which represent the reduction in the number of organisms found in stool cultures and the reduction in the total number of positive individuals found by daily stool cultures are roughly similar for each given organism. It should be noted, however, that the average colony count declines more rapidly than does the total number of detected infected individuals. These graphs are representative of a much larger series of outbreaks studied. From our experience in these studies we now know Flexner infections clear up rapidly and usually completely; Schmitz infections respond only a little less favorably; and that a Sonne infec-

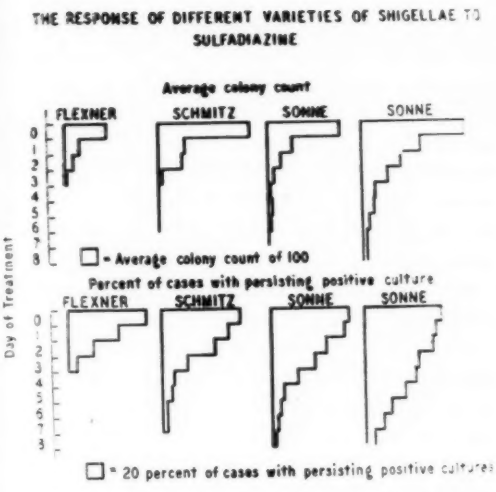

Chart V. The response to sulfadiazine therapy of different varieties of *Shigella* in observed outbreaks as indicated by the percentage of continuing positive cultures and the average plate colony count given by these cultures.

tion is much more likely to be resistant to therapy.

Epidemics of course are special occurrences and do not necessarily reflect the conditions found in endemic infections. A study now in progress of endemic cases shows that the results first cited are substantially those which may be expected in general practice. To date in this series only 1 per cent of the Flexner infections have been resistant to sulfonamide therapy. On the other hand, approximately 12 per cent of the cases due to *S. sonnei* have failed to respond to the various sulfonamides used. Most of these resistant patients recovered symptomatically in a short time, and frequently there was a marked reduction in the number of organisms found in the stool culture, which reduced their hazard as sources of infection for others.

Of further practical importance is the size of the dose of sulfonamide which has been found effective. An outbreak of Flexner infections was abruptly terminated by single doses of 2 grams of sulfadiazine given at the same time to all infected and exposed individuals. The optimum dose has not yet been definitely determined, but for Flexner infections it is probably not over 2 grams per day for four days. Such small doses, however, are not only ineffectual in the treatment of Sonne infections but actually promote the development of sulfonamide resistant strains. This has been observed both *in vivo* and *in vitro*.

Cases of acute diarrhea due to *Salmonella* infection are apparently not benefited by sulfonamide therapy. Due to the large number of types found in this group, this statement must be tentative until a greater number of observations can be made. This much, however, can be said with certainty: No member of this group who has been observed with sufficient frequency to permit controlled observations has been significantly affected by sulfonamide therapy.

DIAGNOSIS

It is obvious from these findings that if a patient is to receive the full benefit of present day knowledge, therapy of the diarrheal disorders must be controlled by stool cultures. To do this easily and accurately requires no more training than is involved in routine blood and urine studies if full advantage is taken of the methods now available. The basic procedure would be the same under all circumstances, but the immediate location of laboratory facilities, the type of practice and other conditions call for modifications to fit the practical situation.

Basically the procedure is as follows: The physician or his nurse helper takes a culture by rectal swab when the patient is first seen, plates it directly to SS agar, and sends the inoculated plate to the laboratory for incubation and identification of such pathogens as may be present. Laboratory methods are available which permit reliable identification within 36 hours after the plate has been received. This is entirely feasible whenever laboratory facilities, either private or public, are available in the same town.

Several modifications of this method have been tried out successfully. In the New Orleans Charity Hospital prepared plates of SS agar are kept in the Central Service of wards which have an active admission rate of diarrheal disease. The interne

takes the culture when he examines the patient and sends the labeled plate back to the Central Service. From there it goes to the laboratory.

It is entirely feasible for physicians to keep the plates in their office, plate the cultures as described, and send the plates to the laboratory at the end of the day. That this method is valuable to the clinician is shown by these facts: On the average, reports to the hospital physician are given verbally within 36 hours after admission when swab specimens are taken. When a stool culture is ordered on the chart, to be taken in the usual routine manner, the average time from admission to verbal report is between six and seven days.

When laboratory facilities are not readily available a slightly more difficult but relatively simple procedure may be used. The culture media are all available in dehydrated form. The medium can be prepared in a few minutes, since it is only necessary to add distilled water, bring the mixture to a boil, and pour it into the plates. They may be inoculated as soon as the agar has solidified. The plates are kept for 18-20 hours in an incubator and then examined. Room temperature can be used but it is not recommended, since growth is slower and colony differentiation less satisfactory. Suspicious colonies, which on SS agar are colorless or white, are then fished to a triple sugar iron agar slant. The reaction produced by growth on these slants permits a preliminary identification which, while admittedly incomplete, gives a very good differentiation of the cases into three groups, those due to *Shigellae*, those due to *Salmonellae* and those from which no pathogen is isolated. Such cultures are easily mailable to a central laboratory for complete identification.

In the event that none of these procedures is feasible, there is another possibility. As we have seen, the type of infection in a given area is not necessarily the same as in another area of the same state. However, through the cooperation of the physician with local health authorities, surveys may be conducted which will show the prevailing type in specific localities and thus permit a more rational therapy.

MANAGEMENT OF CASES

What then, is the method of choice in the management of the acute diarrheas today? No single treatment schedule can be laid down as optimum. The known facts should be adapted to the particular conditions and type of practice of the individual physicians.

Since *Shigella* infections predominate in most areas in this country the following procedure is recommended for hospitalized cases. After taking a culture on admission, sulfadiazine should be started in a dosage of 4 grams per day for adults and proportionately smaller amounts for children. This dosage should be continued if Sonne infection is found, but half this amount is sufficient for Flexner infections. The sulfonamide should be discontinued if *Salmonella* or negative findings are reported. Cultures are then taken on the second or third hospital day and thereafter daily until two consecutive negatives have been obtained. The drug may then be discontinued even though symptoms have not entirely disappeared since a longer time may be required for epithelialization of the extensive ulceration of severe cases than is needed for bacteriologic cure. In New Orleans and other areas in which a high proportion of the cases are due to *Salmonella* infection, it seems better, except in critically ill patients, to wait until an etiologic diagnosis has been made before beginning specific therapy.

In office or home practice, even though an admission culture is feasible, repeated cultures to determine when therapy may be discontinued are usually impractical. A convenient rule of thumb is to treat Flexner infections for five days and Sonne infections seven to eight days.

When no laboratory control is possible, a therapeutic trial with a sulfonamide drug is certainly indicated in all severe cases of acute diarrhea. Treatment should be continued for two days after symptoms have subsided or for a maximum of eight days. The latter limit is set because in our experience infections which are not eradicated by this time have not been benefited by addi-

tional treatment with sulfonamides. Furthermore, when symptoms persist for this length of time without definite improvement, it is quite probable that some etiologic agent other than *Shigella* is responsible for the trouble. Two patients with a bloody diarrhea seen within the past year are illustrative. Both were proved to have *Shigella* infections on admission, both were treated and cleared up bacteriologically, but symptomatically both showed little improvement. In each case, trophozoites of *Endamoeba histolytica* were also found and amebicidal therapy was then effectively used. Apropos of these cases, when treatment is attempted without adequate laboratory control it is well to remember that the great majority of *Shigella* and *Salmonella* infections present a self-limited clinical picture, and the longer the illness has persisted before the patient comes to the physician, the more likely it is that other etiologic agents are responsible.

SUMMARY

It has been shown that the acute diarrheal diseases seen by the physician are usually due to either the *Shigella* or the *Salmonella* group of bacteria. These infections cannot be differentiated clinically from each other or from those due to other agents which can produce similar symptoms. Such a differentiation is of practical importance since sulfonamide therapy is highly effective in shigellosis but is valueless at present in salmonellosis. Since all sulfonamides are potentially dangerous, their use should be restricted to cases with positive indications and they should be given in as small a dosage as is compatible with optimum therapeutic results. Simple but effective bacteriologic controls of therapy are available and await only the interest of the practicing physician before they are put into much greater use.

BIBLIOGRAPHY

Cooper, Merlin L., Furculow, M. L., Mitchell, A. Graeme, and Cullen, Glenn B.: The relation of dysentery to the acute diarrhea of infants and children, J. Pediat., 15:172, 1939.

Davidson, W. C.: Bacillary dysentery in children, Johns Hopkins Hosp. Bull., 31:255, 1920.

Flexner, Simon, and Holt, L. Emmett: Editor's Bacteriological and Clinical Studies of the diarrheal diseases of infancy with reference to *B. dysenteriae*. The Rockefeller Institute of Medical Research.

McGinnis, G. Foard, McLean, A. L., Spindle, F., and Mascy, K. F.: A study of diarrhea and dysentery in Henrico Co. Virginia, Am. J. Hyg., 24:552, 1936.

TenBroeck, C., and Norbury, F. G.: *B. dysenteriae* as a cause of infectious diarrhea in infants, Boston M. & S. J., 174:785, 1916.

Hardy, A. V., and Watt, James: Studies of the acute diarrheal diseases XII. Etiology, Pub. Health Reports, 60: No. 3, Jan. 19, 1945.

DISCUSSION

Dr. Joseph S. D'Antoni (New Orleans): Because of the extreme importance of the diarrheal diseases in both public health and general practice, I want to begin by saying that physicians in Louisiana, and particularly in New Orleans, are fortunate to have had Dr. Watt working among them for the past two years. During this time he has laid the groundwork for the accurate diagnosis and intelligent management of these conditions. Certain points he had made in the course of his presentation tonight are well worth stressing again:

1. The great majority of all cases of acute diarrhea never reach the physician but are managed by home measures or on the advice of the druggist. Patients with acute diarrhea who consult a physician are usually seriously ill.

2. Bismuth and paregoric are sometimes useful in controlling symptoms in diarrheal diseases, but they do not attack the basic pathology. Etiologic diagnosis should be the first step in every case, and therapy should be derived from the diagnostic results.

3. If diarrhea proves to be of *Shigella* origin, therapy need no longer be empiric, for sulfadiazine has been proved to be of value in these circumstances. This fact is of particular importance in pediatric practice because *Shigella* diarrheas in very young children are associated with a high fatality.

4. Sulfadiazine, however, is not the solution in all diarrheas, nor is its routine administration the solution even in *Shigella* diarrheas. It is not the solution in *Salmonella* infections, the incidence of which in the New Orleans Charity Hospital, as Dr. Watt has shown, is approximately 25 per cent. Nor is it the solution in amebiasis, which is likely to be the cause of acute diarrheal manifestations in a large proportion of adults. Finally, since all *Shigella* diarrheas are not due to the same strain, the dosage of the drug and the duration of treatment depend upon the particular strain present in a particular case. Flexner infections usually respond rapidly to sulfonamide therapy, but Sonne infections are frequently resistant and treatment must be carried out over a longer period of time.

In other words, without an etiologic diagnosis, the empiric administration of sulfadiazine in acute diarrhea may mean that a patient is being treated uselessly because he has a *Salmonella* infection;

or that he is being treated for a bacterial disease when actually he has a protozoal infection due to *E. histolytica;* or that he is not being treated long enough because he has a resistant Sonne shigellosis instead of a responsive Flexner shigellosis.

The method of cultural diagnosis which Dr. Watt has outlined is a simple, inexpensive and accurate procedure, and therefore very useful in general practice. On the other hand, I think there is general agreement that the greater the number of examinations, the higher is the percentage of positive diagnoses. For that reason, in my own practice I employ the following more elaborate method:

When I am consulted by a patient with acute diarrhea I request that he collect a specimen of the next stool. He next takes an enema consisting of a quart of physiologic salt solution, and collects a specimen of the evacuated fluid. Finally, he is sigmoidoscoped, at the office or at his home, depending upon the severity of his illness, the bowel is inspected, and material for examination is aspirated directly from the intestinal mucosa. All three specimens are examined for *E. histolytica* and other pathogenic protozoa, and the enema specimen and the material aspirated from the bowel wall are cultured.

The advantages of this method are threefold: (1) All specimens examined are freshly passed or collected, which, incidentally, is one of the advantages of the Hardy-Watt swab method; (2) by inspection of the mucosa it is possible to demonstrate the presence and degree of ulceration and edema, and thus to grade the disease as to severity. The information is less useful when the patient is an infant, but in general it is valuable from the standpoint of prognosis and indicates the course which the disease is likely to follow. (3) A protozoal infection coexisting with the bacterial infection is not overlooked because it is specially looked for.

After the diagnosis is made, my own method of therapy in shigellosis is identical with that outlined by Dr. Watt, whom I should like to congratulate again for his very excellent work.

Dr. D. N. Silverman (New Orleans): If the acute cases are going to be adequately treated, we who have dealt for many years with chronic bacillary dysentery will have to go out of business. However, in the course of events during the past year we have seen more cases of chronic dysentery due to bacillus paratyphosus B, than ever before. Dr. Watt's paper deals only with acute bacillary dysentery, but since chronic bacillary dysentery so commonly follows acute shigellosis, it should be mentioned here. If we do not see these cases in the acute stage, giving adequate treatment, we are going to see many of them as cases of chronic dysentery bacillus infection of the bowel. In these cases of chronic infection, instead of there being superficial ulceration of the bowel, the organism is buried in the walls and causes deep inaccessible infection, pitted scars and pustules. I do not think any drug has been found to dislodge them from their emplacement in the bowel wall. It is also our experience that sulfonamides have no benefit in chronic Salmonella infection of the bowel. It is interesting that many of these cases which have chronic bacillus paratyphosus B infection have given no history of paratyphoid fever and no history of acute dysentery, but started insidiously and became chronic, in some cases over periods varying from three months to six years.

Dr. Watt (In closing): I would like to thank Drs. D'Antoni and Silverman for their remarks and to add a short comment on two phases of the discussion.

The use of multiple specimens as described by Dr. D'Antoni will give a greater number of positive results in cases than will single cultures. The rectal swab technic is not a substitute for the method he uses but rather a simple procedure which provides reliable diagnostic information in a high percentage of the cases of acute diarrhea seen by the physician. These methods of diagnosis are related to each other in much the same way as a simple sputum examination is related to the more elaboate technic of bronchoscopy. The simple examination is frequently an indication for the more complex.

Dr. Silverman's finding of a large number of *Salmonella paratyphoid B* infections is very interesting. To date, we have isolated some 26 different *Salmonella* types from the cases seen at Charity Hospital. The predominating type has varied from time to time. Early in the study *S. newport* was the most commonly encountered type, at present *S. panama* is being isolated most frequently. The clinical significance and the epidemiologic importance of the various types is not well known at the present time. About all that has been established with certainty is that some types are encountered more frequently in acute diarrheal disorders, while others tend to invade the blood stream and give a typhoid-like picture.

In closing, may I express my appreciation to you, the members of the Orleasn Parish Medical Society, for the privilege of presenting this paper at your meeting.

COMPARISON OF INCIDENCE AND TREATMENT OF MENINGITIS OVER A TEN YEAR PERIOD*

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This study was carried out in an effort to evaluate changes and progress in the treatment of meningitis since the advent of the sulfonamides in particular and, by comparison of mortality rates, duration of fever in convalescence, sequelae, and complications, to show what difference our present methods of therapy have made in meningitis. The material used is from the case records of Charity Hospital of Louisiana at New Orleans and the years of 1933 and 1943 were chosen for the study so that progress over a ten year period could be studied, thereby providing a comparison of two periods not too far apart, in which the disposition of the cases was similar except for the to be evaluated alterations in plan of treatment. Although the expected changes derived from the use of the therapeutic measures available in 1943 lay in the treatment of the septic meningitides, for completeness of the study, all cases were included where a reasonably well proved meningitis, septic or not, existed. Where the etiology was determined, the meningitis is classified under a specific etiology; the remainder are grouped as undifferentiated meningitides. Many of these latter possessed features suggesting a specific etiology, but unless the organism was identified bacteriologically or the course (as in lymphocytic choriomeningitis) was unmistakable, or the diagnosis was proved pathologically, that case is included in the undifferentiated group.

In reviewing the case records, a considerable difference in the incidence of meningitis in general as well as in the specific types of meningitis was observed. There were 55,437 admissions to the hospital in 1933 and in only 53 of this number was a diagnosis of meningitis made, whereas, in 1943 there were 42,105 admissions to the hospital and in 139 of this number a diagnosis of meningitis was made. The following table (table I) has been prepared to show the comparison of incidence in the years studied showing the number of cases occurring and their percentage relationship to the total number of admissions for that year.

TABLE 1

	1933		1943	
Total admissions	55,437		42,105	
	Number of cases	Per cent of total admissions	Number of cases	Per cent of total admissions
Total cases of meningitis	53	.09	139	.33
Meningococcal	20	.035	53	.125
Pneumococcal	6	.010	25	.059
Influenzal	3	.005	17	.0406
Tuberculous	2	.0035	16	.039
Staphylococcal	2	.0035	4	.0095
Streptococcal	7	.012	1	.0023
Torula	0	0	1	.0023
B. pyocyaneus	1	.0017	0	0
Choriomeningitis	0	0	2	.0046
Aseptic (post encephalography)	0	0	1	.0023
Luetic	0	0	1	.0023
Undifferentiated	12	.020	18	.042

In table 1 the increase in the incidence of meningitis in general is shown. The total cases of meningitis admitted to the hospital in 1933 comprised only 0.09 per cent of the total admissions to the hospital while in 1943, meningitis accounted for 0.33 per cent of the total admissions. This table also shows the alterations in the incidence of the specific types of meningitis, but it is better shown in table 2, where the specific types are compared to the number of cases of meningitis.

Table 2 presents the data in regard to the incidence of the various types of meningitis showing the percentage relationship of the incidence of the specific types of meningitis to the total number of meningitis cases for that year.

The data in table 2 show that in regard to special types, the relative incidence of meningococcal meningitis to be not significantly altered; but in marked contrast one

*Read before the Orleans Parish Medical Society, March 12, 1945.

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TABLE 2

	1933		1943	
Total cases of meningitis	53		139	
Specific types	Number of cases	Per cent of total admissions	Number of cases	Per cent of total admissions
Meningococcal	20	37.7	53	38.4
Pneumococcal	6	11.3	25	18.3
Influenzal	3	5.6	17	12.2
Tuberculous	2	3.77	16	11.7
Staphylococcal	2	3.77	4	2.9
Streptococcal	7	12.45	1	.719
Torula	0	0	1	.719
B. pyocyaneus	1	1.88	0	0
Choriomeningitis	0	0	2	1.4
Aseptic	0	0	1	.719
Luetic	0	0	1	.719
Undifferentiated	12	22.6	18	12.09

notes a great increase in the number of cases of pneumococcal, influenzal, and tuberculous meningitis. Before attempting to evaluate this increase, the decrease in percentage of the undifferentiated types of meningitis over the ten year period must be explained. The improvement in diagnostic methods relative to pneumococcic and influenzal meningitis may be expected to have facilitated more accurate diagnosis; however, little change in the effectiveness of diagnostic determinations in tuberculous meningitis were apparent. Thus one can reasonably ascribe at least part of the increase in two of the three mentioned specific types to improvement in diagnostic methods. The decrease in the relative number of cases of staphylococcic and streptococcic forms of meningitis is evident.

Since these latter varieties were found to be so often secondary manifestations, it may be offered that not only are sulfonamides of therapeutic value in septic meningitis but perhaps they also have served to decrease the incidence of certain types. This premise, however, is weakened by the finding of an increase in pneumococcal meningitis which also, though much less frequently in 1943, occurred at times as a secondary manifestation.

One fact not mentioned in the tables, but of possibly considerable importance, is the finding that all but one of the 17 cases of influenzal meningitis occurring in 1943 were found to be caused by *H. influenza* Type B. In the one case making the exception, the organism was unfortunately not typed. The three cases of influenzal meningitis occurring in 1933 were not typed. All the cases of tuberculous meningitis included in this series were proved at autopsy, a characteristic basilar meningitis being the rule, but a coincident tuberculoma being found in one case in 1933. No sound clinical criteria for the diagnosis of tuberculous meningitis could be found in this study. Chloride determinations varied so widely as to be useless; Mantoux tests were sometimes negative in definite cases proved at postmortem. Levinson's test was negative in some proved cases of tuberculous meningitis and positive in some cases later proved to be non-tuberculous. In most of the cases of tuberculous meningitis, the meningeal involvement was but part of a picture of miliary tuberculosis.

After considering the various incidences presented above, the most obvious way of evaluating the effect of the therapeutic methods in 1943 compared to those used ten years previously seemed to be by studying the percentage of fatalities for the two periods. This was done, and the findings are presented in table 3.

It may be seen from table 3 that the percentage of deaths due to all types of meningitis decreased from 79.3 in 1933 to 49.7 in 1943 changing the expected survival rate from one in five in 1933 to one in two in 1943. It is to be noted also that these figures are compiled using not only septic meningitis but using tuberculous meningitis as well. The latter, in fact, was the cause for 16 of the 69 total deaths due to meningitis in 1943.

If the cases of tuberculous meningitis are excluded from the compilation, the percentage of deaths due to all other types of meningitis is found to be 76.9 in 1933 and the corresponding figure for 1943 to be 43.8. Even this latter figure is shocking, but upon breaking the total down and analyzing each type of meningitis separately the findings are more encouraging. It was found, for instance, that the percentage of deaths in meningococcal meningitis was reduced from

TABLE 3

	1933					1943				
	Total Cases	Recovered No.	Recovered Per cent	Died No.	Died Per cent	Total Cases	Recovered No.	Recovered Per cent	Died No.	Died Per cent
Meningococcal	20	6	30	14	70	53	44	83.01	9	16.99
Pneumococcal	6	0	0	6	100	25	7	28	18	72
Influenzal	3	1	33⅓	2	66⅔	17	5	29.3	12	70.7
Tuberculous	2	0	0	20	100	16	0	0	16	100
Staphylococcal	2	0	0	2	100	4	1	25	3	75
Streptococcal	7	0	0	7	100	1	0	0	1	100
Torula	0	0	0	0	0	1	0	0	1	100
B. pyocyaneus	1	0	0	1	100	0	0	0	0	0
Choriomeningitis	0	0	0	0	0	2	2	100	0	0
Aseptic	0	0	0	0	0	1	1	100	0	0
Luetic	0	0	0	0	0	1	0	0	1	100
Undifferentiated	12	4	33⅓	8	66⅔	18	9	50	9	50
Total	53	11	20.7	42	79.3	139	70	50.3	69	49.7

70 in 1933 to 16.99 in 1943, and that the 100 per cent mortality in pneumococcal meningitis in 1933 was reduced to 72 per cent in 1943. The number of cases reviewed in this series related to the less frequent types of meningitis is too small to allow conclusions to be drawn from their separate indications.

In considering the large number of deaths occurring in both the years studied, it should be noted that many of these patients were in extremis on admission and had been ill often for many days before being brought to the hospital. Several of the deaths in both years occurred while the patients were in the process of being admitted to the hospital and before any treatment was instituted. Such instances may detract from the accuracy of the data regardings deaths due to specific types but for the group as a whole the error should be fairly well equalized, giving an accurate relationship between the two periods although leaving the percentage of deaths at a somewhat greater number for both years than should have occurred otherwise.

Aside from comparing the percentage of deaths for the two periods, another criterion to aid in evaluating effects of more recent therapeutic measures was the comparison of the duration of illness in the cases which survived.

Tables 4 and 5 were prepared to show the duration of fever during convalescence, counting from the day of admission to the hospital, since this was, as a rule, the day when treatment was begun.

TABLE 4
1933

Type of Meningitis	Minimum number of days	Maximum number of days	Average number of days
Meningococcal	7	33	15.5
Influenzal	10	10	10.0
Undifferentiated	5	18	11.0

TABLE 5
1943

	Minimum number of days	Maximum number of days	Average number of days
Meningococcal	0	16	4.3
Pneumococcal	2	14	4.6
Influenzal	2	54	20.5
Staphylococcal	6	6	6.0
Choriomeningitis	4	4	4.0
Aseptic	16	16	16.0
Undifferentiated	.5	6	3.4

In this comparison, only the data regarding meningococcal, influenzal, and the undifferentiated varieties are relevant, since only in these three did any recoveries occur in 1933; the other data are presented only incidentally.

The striking difference in the duration of fever in meningococcal meningitis for the two periods shows only partially the remarkable difference in the response of the patients to treatment. Whereas, in 1933 convalescence was at best a stormy, trying, pessimistic, and often unexpected period, a characteristic response with comparatively

amazing improvement in the condition of the patient was the expected rather than the unusual in 1943.

When considering the actual methods of therapy used during these two periods it was found that only one worthy weapon was available to the clinician in the treatment of meningitis in 1933. This, of course, was specific antiserum. Antimeningococcal serum was extensively used, not only as the only available agent against the meningococcus; but, showing the desperation of the therapeutist, it was even used when the etiology of the meningitis was known to be of another type, such as streptococcic for instance where five of the seven cases in 1933 received antimeningococcal serum, naturally with no benefit. Pneumococcal antiserum must have been poorly established or seldom available in 1933, since only one of the six cases (a Type III pneumococcus found) received specific antiserum, and this patient died. Of the three cases of influenzal meningitis occurring in 1933 only one received anti-influenzal serum, and this was the only case surviving. Another evidence of the feeling of futility that accompanied the treatment of meningitis in 1933 was the notation in two cases of the use of injection of Pregl's iodine and acriflavine respectively into the carotid arteries bilaterally and simultaneously, the carotids being exposed surgically. The fatal outcome was not apparently influenced in either case. Staphylococcal antitoxin was used in a case of influenzal meningitis thought to be due to staphylococcus because of the concurrent presence of acute osteomyelitis; this case also expired.

The number of serum reactions occurring in 1933 was studied and it was found that 33 patients received antimeningococcal serum and that nine of these (27.2 per cent) had serum reactions varying in severity from those of anaphylactic type nearly causing death, to mild serum sickness occurring during convalescence. In one case, the death of the patient was attributed to the administration of serum, resulting in a fatal reaction in spite of negative sensitivity tests. The total percentage of serum reactions from antimeningococcal serum was found to be 27.2. No untoward reactions followed the use of antipneumococcic, anti-influenzal, or antistaphylococcic serum (each was used only once).

In contrast to the picture in 1933, it was found that only ten years later serum was not used alone in a single case. A number of patients did receive serum, of course, but always as adjuvant therapy to sulfonamides. The "repeated tap" method that was used on all patients a decade ago, was used only very occasionally in 1943. Often, in fact, no further spinal taps were done after the initial diagnostic one and, thus, what previously was done as both a diagnostic and therapeutic procedure has become largely a diagnostic procedure. One sees no more a chart recording 40 spinal taps under "therapy" on a meningitis chart.

The only cases of meningitis occurring in 1943 in which sulfonamides were not used, were three cases of tuberculous meningitis, one case of luetic meningitis, and those cases in which the patient died before therapy could be instituted. Sulfadiazine was by far the most widely used drug in this series, although several other sulfonamides were used occasionally. Table 6 shows the relationship of the various drugs in respect to the number of cases applying.

TABLE 6

	Sulfadiazine		Sulfathiazole		Sulfapyrazine		Sulfapyridine		Sulfamethazene	
	Lived	Died	Lived	Died	Lived	Died	Lived	Died	Lived	Died
Meningococcal	37	8	4	0	2	0	1	0	0	0
Pneumococcal	7	9	0	5	0	2	0	0	0	1
Influenzal	4	10	1	0	0	2	0	0	0	0
Tuberculous	0	10	0	1	0	1	0	1	0	0
Staphylococcal	1	2	0	1	0	0	0	0	0	0
Streptococcal	0	1	0	0	0	0	0	0	0	0
Torula	0	1	0	0	0	0	0	0	0	0
Choriomeningitis	1	0	0	0	0	0	0	0	0	0
Aseptic	1	0	0	0	0	0	0	0	0	0
Luetic	0	0	0	0	0	0	0	0	0	0
Undifferentiated	7	6	0	2	1	0	1	0	0	0
Total	58	47	5	7	3	5	2	1	0	1

No attempt was made to draw conclusions regarding special value of the specific drugs from table 6 since the number of cases receiving other than sulfadiazine was so small by comparison, but the table shows well the pre-eminence of sulfadiazine as the princi-

pal sulfonamide used in these cases.

The next point inviting study was the number of reactions to the sulfonamides. Of 129 patients receiving sulfonamide therapy, 20 patients had untoward reactions to the drug used (15.5 per cent). Only five types of untoward reactions were observed, namely: anuria; drug eruption, hematuria, drug fever, and agranulocytosis. In 105 patients receiving sulfadiazine, there were 13 untoward reactions (12.3 per cent) and, of these, nine consisted of hematuria occurring in acid urine, two of hematuria in alkaline urine, and two of so-called drug fever. One of the nine cases of hematuria in acid urine developed anuria, but this latter was overcome. Two untoward reactions were observed following the use of sulfathiazole out of a total number of twelve cases (16.6 per cent) and of these one had hematuria in acid urine and one had so-called drug fever, one other patient developed a drug rash after changing from sulfadiazine to sulfathiazole. The one case of agranulocytosis occurred following the use of sulfapyrazine and promptly recovered after discontinuing the drug and institution of proper treatment for granulocytopenia. Two reactions were seen following the use of both sulfadiazine and sulfathiazole in the same patients and both of these reactions consisted of hematuria in acid urine. Two similar reactions occurred following the combined use of sulfadiazine and sulfanilamide. It is of interest that no deaths in this study were attributed to untoward reactions to sulfonamides and in only a very occasional instance was there a serious enough reaction to require discontinuance of the drug. Also of interest is the finding that of the 15 cases of hematuria, only two (13.3 per cent) occurred when the urine was alkaline and even in these two there was no evidence to show that the urine was alkaline at the time the actual damage was done. No direct correlation between blood level of the sulfonamide and untoward reaction could be drawn.

Hematuria also occurred with blood levels as low as 4.6 mg. per cent and was absent in patients with as high a level as 46 mg. per cent. The sulfonamide level in the case of agranulocytosis was 3.1 mg. per cent, and in the cases of drug fever the drug level averaged 11.4 mg. per cent. The single case of anuria occurred with a sulfadiazine level of 29.5 mg. per cent.

No longer was antiserum or antitoxin found to occupy the pre-eminent position as the most potent therapeutic force against meningitis, but it still occupies a place of importance as an adjunct to sulfonamide therapy. The use of serum in 1933 has already been discussed and its singular importance verified. In 1943, serum was used in 13 of the 53 cases of meningococcic meningitis and of these 11 lived and two died. Fourteen cases of pneumococcal meningitis received serum and of these five lived and nine died, but one must remember that only seven cases of pneumococcal meningitis recovered. Eight patients with influenzal meningitis received serum and of these four lived and four died, but here too one must consider that only five persons with influenzal meningitis survived. Serum was used in two cases of undifferentiated meningitis (one case received pneumococcal antiserum and one meningococcal antitoxin) because of certain clinical features of these cases. Both recovered, but the etiologic agent of the meningitis was never determined. The only case of staphylococcal meningitis to recover received staphylococcal antitoxin in conjunction with sulfonamide therapy. Table 7 shows the distribution of the cases receiving specific antisera.

TABLE 7

	Recoveries	Recoveries receiving serum	Fatalities	Fatalities receiving serum
Meningococcic	44	11 (25%)	9	2 (22.2%)
Pneumococcic	7	5 (71.4%)	18	9 (50%)
Influenzal	5	4 (80%)	12	4 (33.3%)
Total	56	20 (35.7%)	39	15 (38.5%)

From these data it is apparent that specific antisera is of definite value in the treatment of influenzal meningitis, since 80 per cent of the recoveries received antiserum and only 33 1/3 per cent of the fatalities received it; one may also accept the value of pneumococcic antiserum on similar

but less pronounced relationship. The value of antimeningococcic serum (actually meningococcic antitoxin) is not so well shown, but one must remember that peculiar difference which a compilation of data cannot show, that is, the fact that only the most desperate cases of meningococcic meningitis received antitoxin, and with this consideration in mind, one is justified in believing that meningococcic antitoxin is of definite value when used within its well understood limitations and in proper amounts.

Other methods of therapy for meningitis that presented were: namely, "promine", as used in two cases of tuberculous meningitis, without effect; bismuth, used successfully in one case of luetic meningitis, and in the case of torula meningitis, iodides, bismuth, and arsenicals, without effect. Two patients in this study were given penicillin in conjunction with sulfonamide therapy; one of these patients who had an undifferentiated meningitis survived, and the other patients who had infection with pneumococcus failed to recover.

In studying the serum reactions occurring in 1943 it was observed that of 38 patients receiving serum, 14 had reactions, giving a rate of 36.8 per cent for serum reactions in 1943 as compared to 27.2 per cent in 1933. Of the serum reactions in 1943, nine of the total were due to meningococcal antitoxin, four were due to antipneumococcic serum, and one questionable reaction was due to anti-influenzal serum. None of the serum reactions in 1943 resulted fatally and in general they seemed less severe than those occurring in 1933.

The complications occurring in patients with meningitis were tabulated and it was noted that in 12 of the 53 patients studied in 1933, complications occurred which could rationally be thought related to the primary disease. Oddly, pneumonia was diagnosed (or recorded) in only one case premortem though it was found in a large percentage of the cases at autopsy. Distention was cause for concern in three cases, mastoiditis in one case, sinusitis (unspecified) in two, otitis media in four, and cranial osteomyelitis in one case. Of the 139 cases studied in 1943 it was found that 20 cases had related complications. Nine of these patients were diagnosed as having pneumonia, and of these one had a pleural effusion. One patient developed pulmonary infarction; one had diabetic acidosis, one a gluteal carbuncle, one was ascribed to have nephritis (non-sulfonamide) but the record is inconclusive. Seven cases of otitis media as a complicating factor were observed.

SEQUELAE

A study of sequelae of this group of diseases was attempted. Unfortunately the follow-up of such a group is generally very poor as would be expected by the very nature of the disease and, therefore, this study is recognized to be deficient. Nevertheless, the findings will be presented. The 1933 records especially were inadequate, since clinic records were seldom available and in that group the only sequelae recorded were of three cases of polyarthritis and three cases of persistently positive nasopharyngeal cultures for meningococcus. In 1943, the clinic records were more readily obtainable and record was found of 15 cases where sequelae occurred. The following is a list of the sequelae which were recorded:

Partial paralysis of left deltoid and supraspinatus muscles.

Bilateral deafness; polyarthritis after a serum reaction.

Unilateral seventh and eighth nerve palsy.

Hallucinosis and euphoria.

Deafness, bilateral; and cerebral atrophy (proved by encephalogram).

Joint pains and profuse sweats (no serum used in this case).

Polyarthritis (no serum used in this case).

Weakened ocular convergence.

"Bad child"—evident behavior disorder, which eventually was relieved.

Burning of eyes for weeks.

Polyarthritis (no serum given to this patient).

Partial left hemiplegia with paralysis of left deltoid.

Stupidity (previous state uncertain).

Sixth nervy palsy.
Corneal ulcer.

SUMMARY

In an effort to study the effects of sulfonamide drugs in the treatment of meningitis, all cases of meningitis in the Charity Hospital in 1933 were compared with all cases of meningitis occurring in 1943. No selection of cases was made other than to avoid inclusion of cases where the diagnosis of meningitis was unsatisfactorily proved. Meningitis was classified etiologically and those cases where the etiologic agent was not proved, were grouped under "undifferentiated meningitis." The incidence of the various types was presented, the percentage of deaths in the various types was compared for the two years. The incidence of meningitis in general was shown in its relationship to the total number of admissions to the hospital. The period of convalescence was compared. The therapeutic methods were presented briefly and a comparison of results and untoward reactions to the therapeutic agents shown. The complications of meningitis during both periods were presented as were the sequelae. As much of the data as possible were presented in tabular form and brief supplementary remarks were made where the tables were not self-explanatory.

DISCUSSION

Dr. P. H. Jones: Meningitis some fifty years ago was spoken of in two groups: meningococcal, that meningitis sometimes got well; the other types did not. It was seen from the doctors' interesting paper that we have moved a long way in the field of treatment since then.

Dr. John H. Musser: Mr. President, to my mind there are two things of paramount interest in this report which we presented to you tonight. In the first place I am unable to explain the increasing incidence of meningitis. You will note that not only has meningococcic meningitis increased in the actual number of cases but also that other types of meningitis have also increased. Dr. Rollings has suggested, in talking these things over with me in preparing the paper, that this might be due to the fact that at the present time we are keener in our diagnostic acumen and able to recognize cases of meningitis sooner than we were able to do ten years ago. I doubt this very much. I think you will appreciate and realize, those of you who have worked in the contagious wards in Charity, that we were just about as ready to do lumbar punctures then as at the present time. The puncture is essential to make the diagnosis; as my old professor once remarked—"the diagnosis flows out of the needle," which is perfectly true. Of course in wartime for some peculiar reason, meningitis is on the increase not only in army camps but also in the civilian population. I can see very well why meningococcic meningitis is on the increase but can not explain the reason pneumococcic meningitis has increased or why there are more cases of tuberculous meningitis.

The second thing which I think of decided importance is the question of the sulfonamides in the treatment of meningitis and more particularly the effects of penicillin. As Dr. Rollings pointed out, we have not had very much opportunity of using penicillin. We have used it more in the past year since it has become available than we did in 1943 but I do not think the results are a bit more satisfactory than they are with sulfonamides. As a matter of fact, in an article written by Dr. Francis Blake published in last week's J.A.M.A., he said the sulfonamides are better drugs than penicillin in meningococcus meningitis. I think that statement goes with our ideas about the treatment of this disease.

In adults I recommend that the patients be given sulfonamides by mouth or intravenously if they can not be taken by mouth. Practically all the patients take the drug by mouth after the first forty-eight hours. Then give "booster" doses of penicillin intrathecally if there is not a satisfactory response to sulfonamides. The penicillin works so beautifully when applied locally in septic conditions I think it helps materially in clearing out organisms in the spinal fluid.

Our recovery figures are not quite as dramatic or remarkable as some from the army camps. On the other hand I hope you will realize and appreciate many of the patients we get at Charitp Hospital are *in extremis* when they come into the hospital. In army camps as soon as a boy has a headache or feels badly he reports to the medical officer, the battalion surgeon. Their cases are seen much earlier than ours.

Our results in the past were poor as contrasted with other places. About the time of this analysis of the first ten year period Dr. Tripoli read a paper before the A. M. A. section on medicine upon the treatment of meningitis. We were rather criticized because our mortality rate was much higher than in, say New York City. I might say, in extenuation, that in New York City the City Board of Health at that time had a meningitis department, Dr. Josephine Neal heading it. Irrespective of the patient's economic place in life, it helped the private physicians with their patients who received treatment early and received it more definitely and positively, more thoroughly and more properly than would the patient being seen

on the outside here in New Orleans and the surrounding districts.

The recent results of treatment have been so remarkable that I have heard it said among the laity that meningitis now is not as bad as having a bad cold. I will say, however, that within the last several months we have had three or four very typical fulminating cases with marked delirium, death occurring in eighteen to thirty-six hours. I have a sort of idea, which may be wrong, that in so far as meningococcic meningitis is concerned, the present strain is not as virulent as in the past. There is a possibility that it may become more virulent and that the fulminating cases now appearing suggest this possibility.

One last remark—I think that it is a good idea to give antitoxin to patients promptly. When patients come in markedly prostrated, sometimes wildly delirious, sometimes in coma, if given one hundred thousand units of antitoxin intravenously and then followed several hours later by sulfadiazine, better results will be achieved than achieved in the past. We have not made this a routine. I think it is something to which we should give serious thought, as well as the use of penicillin in conjunction with sulfa drugs.

Dr. Gilbert C. Anderson: Mr. President, gentlemen, I have nothing of value to add. This is largely reminiscent with me because I was probably responsible for the injection of the iodine preparation and the dye stuffs directly into the carotid arteries. I remember we did on several patients in Charity and at the Nose and Throat Hospital with the late Dr. Lynch with no apparent result on the progress of the disease. Just here of late I have been unfortunate enough to be associated with two cases of rather fulminating pneumococcic meningitis that did not respond to intensive treatment either with chemotherapy or molds or, as a matter of fact, with a combination of both; even to the point of treatment by cisternal puncture. It has been given, as you know, in a few instances by direct installation into the ventricles but I saw lately reported very severe reactions following that particular type of therapy with a note of warning sounded against it. The most remarkable difference that I can see is at the Ear, Nose and Throat Hospital—I have been on the staff there a long time and often used to be called when they had intracranial complications from sinuses, mastoid and middle ear and so forth, probably several times a month. I don't believe I have been called down there five times within the past year which I attribute largely to new type of therapy.

That one fact emphasizes very well the contrast between 1933 and 1943 in therapy of intrathecal suppuration.

Dr. R. V. Platou: I would like to suggest that the increase of meningitis in wartime is apparently due to a rapid shift in population. This is shown by increased numbers of carriers and cases when new recruits come into camps or aboard ships. With such migrations there is almost always an increase in carrier rate and incidence of clinical streptococcal and meningococcal infection. This increase is not easy to explain but is probably related to crowding, agitation, shifting, and mixtures of population which do not take place in peacetime.

I would like to say a few words about intrathecal penicillin. We have agreed that the results of treatment with sulfonamides and specific antisera are as good or better than with penicillin except in two particular situations: when the case of meningitis is due to sulfonamide-resistant strain of meningococcus or when pneumococcal meningitis occurs in an infant or young child. With specific serum-sulfonamide treatment the mortality rate for pneumococcal meningitis in young children under three years of age still approaches seventy per cent. We have used intrathecal penicillin in several of these cases, giving it at intervals of twelve and twenty-four hours in doses of twenty to forty thousand units. While it is too early to say what the eventual prognosis will be with penicillin, the results seem to be good so far—better than any yet employed.

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THE EFFECTS OF INADEQUATE ASCORBIC ACID INTAKE

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HISTORY

Scurvy is believed to have occurred among the armies of ancient times. Pliny states that the Roman Army under Caesar Germanicus, after an encampment of two years in Germany, suffered with a disease in which the teeth dropped out and the knees became paralytic. Similar references can be found in the works of Hippocrates, Strato, Celsus, and Galen. However, all the descriptions of this disease offered by these men were too vague to identify them with certainty as scurvy.[1]

The first good description of scurvy is found in the history of Louis IX by Le Sieur de Joinville who wrote of the disease in the army of Christian Crusaders in Egypt in about the year of 1260.[1] However, the first description written by a physician is found in a letter sent in 1541 by one Ectheus to Dr. Blienburchius of At-

recht. The first book completely devoted to scurvy was written by Balurn Ronsseus in 1564.[2]

Numerous accounts are found in the older writings of the ravages of this disease and of the great losses of men which occurred in all long sea voyages. Practically no explorer or historian fails to mention the great death toll claimed by scurvy.

It is very interesting to find, even in the earliest of these writings, with what constant reiteration the juice of lemons or orange, green herbs, brooklime, watercress, and fresh vegetables are described as specific cures for this disease. At the same time it is astonishing that conditions were not improved to prevent this life taking malady, even in spite of the knowledge of a specific cure.[1, 2, 3]

To James Lind, with the collaboration of Sir Gilbert Blane, James Cook, and Thomas Trotter goes the credit for having amassed the greatest amount of convincing evidence as to the nature of scurvy. It was through this influence that the British Navy introduced lemon juice in the rations of sailors as a prevention against scurvy in 1795.[1, 2, 3, 4]

It was not until 1928, however, that Szent-Gyorgii isolated from the suprarenal glands of oxen and from various plant sources the crystalline compound which he called hexonuric acid and is now known as vitamin C, cevitamic acid, or ascorbic acid. In 1932 he proved this compound to be the specific antiscorbutic factor.[8]

Many other names enter into the history of scurvy and ascorbic acid, but a detailed account is beyond the scope of this paper. Reference is made to other sources for a complete history on this subject.

INCIDENCE

Croft and Snorf[5] report the incidence of cevitamic acid deficiency in 100 unselected patients. They found that 38 had plasma ascorbic acid concentrations below 0.40 mg. per cent as determined by the method of Farmer and Ott. The remaining 62 patients had vitamin C concentrations ranging from 0.40 to 1.55 with an average of 0.71 mg. per cent. The lower limit of normal in this study was a plasma concentration of 0.40 mg. per cent.

It is interesting to note that the majority of plasma ascorbic acid concentrations were made on patients who were of an economic and social status that would presuppose a sufficiency of the deserved diet. As a result they concluded that moderate to severe grade of inadequate intake of ascorbic acid is not uncommon among people economically able to obtain a sufficiency of foods which presumably would prevent it. Another interesting observation made during this study was that extremely low plasma concentrations of cevitamic acid may exist without striking clinical manifestations.

In 1942 Baumann and Brook[6] reported the incidence of vitamin C deficiency in European and colored school children of the Cape Peninsula between the ages of eight and sixteen. In determining the deficiency state they used the method of Wright Lilienfeld and MacLenathen for the intravenous saturation test and the method of Gothlin for capillary fragility test. They found that a definite deficiency state was present in 21.1 per cent of European children and 7.4 per cent in colored children. The total number of children examined was 380. These authors conclude that in general there is very little evidence of ascorbic acid deficiency in the children examined.

As a result of the war with its effects on nutrition, there has been an increase of deficiency states in school children, medical students and scientific workers, though the latter two groups have been affected less than the former. However, there has been a decided increase in the incidence and degree of deficiency in the latter groups within the last few years of the war.[7, 8]

There is also a seasonal variation in the incidence of cevitamic acid and deficiency, the levels of plasma ascorbic acid concentrations being low after the winter months and much better after the summer. The basis for making this statement lies in the results of studies[7, 9] carried out on school children, medical students, scientific workers and unselected hospital patients.

PATHOLOGY

The primary morphologic effects of vitamin C deficiency occur in the intercellular substances of certain mesenchymal derivatives. The basic defect is in the formation of fibrils in the intercellular matrix of the fibroblasts. This defect occurs not only in soft tissues but also in osteoid tissue, dentin and it is believed to occur in blood vessels. There is also a tendency for defective materials to form in connective tissue in partial depletion and for nothing of a substantial nature to form in complete depletion of ascorbic acid.[10, 11]

In the capillaries morphologic changes have not been detected and it is doubtful whether they occur. Where the lesion occurs in the capillaries is not known. It may occur in the sheath or in the cement substance between endothelial cells.[11]

The anatomic manifestations of scurvy are greatly modified by two factors, growth and stress. These are of extreme importance to the pathology of ascorbic acid deficiency.[11]

The growth factor and its influence on the morbid anatomy of scurvy was the reason why for many years this disease was considered a variety of rickets. Hematomas became less and less frequent as the age of the patient increased. Osteoporosis is greatly intensified in the young animal just as it is always most pronounced at a particular part of bone where growth is more active.[11]

Stress modifies the site of lesions and determines the extent and involvement of the various structures. Stress plays a major part in determining the site of the hemorrhages and where other changes in muscle will occur.[11]

The lesions are also indirectly modified by other factors, for example the presence or absence of a variety of other diseases and disturbances associated with an increased metabolic rate. These may operate similarly to growth and physical stress in determining the requirements of vitamin C and, therefore, indirectly, the extent and degree of scorbutic lesions.

Skeletal lesions are commonest in the costochondral junctions, the distal end of the femur, the proximal end of the tibia, femur and wrist. In the affected regions bone formation ceases and the existing bony shell becomes rarefied, widened and conical. The epiphysis is not affected in early stages but may become displaced. Microscopic examination reveals a rarefaction of the existing cortex, cessation of bone growth and replacement of the normal junction by a zone of collagen-poor connective tissue in which are embedded fragments of densely calcified cartilage matrix. Frequently these lesions are complicated by hemorrhages, either large subperiosteal ones, or small ecchymosis within or along the bone. The extent of the lesions, of couse, depends on the degree of deficiency, on stress, on growth and on incidental factors.[1, 10,11] Mouriquand and Edel[12] report on the great incidence of decalcification of the neck of the femur which suggests that this location is a place of particularly low resistance to vitamin C deficiency. They consider that this observation is important because of the resemblance of this condition to certain clinical syndromes encountered in children and old people.

In the teeth of adults with scurvy, the dentin is seen to be resorbed and parotic. The little replacement dentin that may be formed is inferior in appearance. The lesions develop first in the apex of the tooth and bifurcation of the root canal. The cementum is similarly affected. Lesions of the gingiva occur only when teeth are present and are most severe about deformed or broken teeth. The gums become boggy, swollen and bleed easily. Rarefaction of the alveolar bones results in the loosening of teeth. The gingival lesions commence on the papillae, first as a hypermia followed by disintegration of the epithelium, infection with ulceration, granulations and even gangrene.[1, 10, 11]

In muscle fragmentation of striated fibers and intense reparative efforts marked by multiplication of the sarcolemma occur. Spontaneous hemorrhages also occur within the muscle. In the eyes, hemorrhagic mani-

festations appear on the conjunctiva, eyelids and elsewhere about the eyes. In the skin the characteristic manifestation is a perifollicular or petechial hemorrhage. These are most common in the lower extremities or wherever pressure exposes the weakness of the capillaries.[10, 11, 12]

Other common lesions are bloody effusions into serous cavities, edema of ankles, enlargement of the heart, and atrophy of bone marrow. The adrenals atrophy as a result of absorption of corporal fat and ascorbic acid. Atrophy of the lymphatic tissue and to a lesser extent of other organs especially glands of internal secretion may occur.[10]

SYMPTOMATOLOGY

The manifestations of ascorbic acid deficiency depend to some extent on the age of the patient. This has already been mentioned above. Infantile scurvy differs from adult scurvy principally in the extent to which the growing bones are involved, but discrepancies in morphology and symptomatology based on the patient's age level do not justify separation of the resulting syndromes into two entities.[3]

The symptoms of this disease begin, as a rule insidiously with a feeling of general weakness and inadequacy, negativism, depression and even melancholia. The normal degree of alertness is replaced by a disposition of inactivity, the patient preferring to sit down or lie about. Anorexia develops which is further enhanced by painful gums. The skin is rough and dry and it may take on a waxy appearance. Soon in addition to the fatigue, there are breathlessness and dull aching pains in the legs and feet. Instead of pain, the patient may complain of stiffness of the knees or feebleness of leg muscles. [1, 3, 14, 15]

The lips become cyanotic, the gums become very red and spongy and bleed upon the slightest provocation. The swelling begins first and is most intense in the lower jaw and usually beginning about the molar teeth and progressing forward. The swollen tissue next ulcerates and breaks down, often sloughing away until the necks of the teeth are left bare. The teeth become loose and often fall out. The breath becomes exceedingly offensive.[7, 14]

The characteristic hemorrhages appear first as petechial spots, later these spots become larger. The spots may be of all colors; red when the effusion first occurs, later becoming purplish, and varying greenish-blue to dusky yellow as they become absorbed. The spots are not painful or tender on pressure unless there is at the same time effusion into muscles. If the disease proceeds untreated there is a tendency for these hemorrhagic areas to ulcerate, particularly when on the legs.[1, 14]

The lower extremities develop swellings in the muscle masses, particularly in the extensors and adductors of the thighs and in the calves, as a result the knees are held in partial flexion; efforts toward full extension are accompanied by great pain. These symptoms are due to hemorrhages into the muscle as well as to under the periosteum. Later a pitting edema will develop which at times involves the whole extremity.[1, 3, 14, 15]

In severe cases there may be hemorrhage from the nose, stomach or intestine, or an extravasation of blood may suddenly appear without provocation at some bizarre site as in the orbit causing proptosis and ecchymosis of the eyelids. Suppuration may develop in any hematoma, leading to the formation of huge abscesses. The pulse may become rapid and weak, and the patient may suddenly die from relatively mild physical exertion.[3]

Infants are usually fretful and take a motionless frog-like position of the lower limbs. Anorexia is prominent. Fever is common in contrast to adults who are as a rule afebrile. Petechial hemorrhages are generally less conspicuous than in the adult. The tenderness of the involved extremities and the changes in contour brought about by subperiosteal hemorrhages and epiphyseal infarction are often extreme. On the other hand, the oval signs are usually limited to swelling and purplish discoloration of the gums.[3, 13]

Anemia is always present both in infants and adults, the picture being that of a secondary anemia due to bleeding. The

leukocyte count may be normal but it is frequently increased and counts from 20,000 to 50,000 have been recorded. The polymorphonuclears are relatively decreased and there is a considerable increase in large lymphocytes and monocytes.[1]

On x-ray the deficiency of intercellular material is manifest in growing bone by lack of matrix or osteid tissue at the diaphysis immediately shaftward from the zone of preparatory calcification. This appears in the x-ray as a zone of diminished density or rarefaction which has become known as the scorbutic lattice. The defective calcification at this zone predisposes to posture and slipping of the epiphysis. At the same time cessation of growth permits an intensification of calcification at the epiphyseal ends of long bones and at the periphery of the epiphyseal centers of ossification which on x-ray appear as the white lines of scurvy. Thinning of the cortex and trabeculae of the shaft gives the bones a ground glass appearance by x-ray. Finally subperiosteal hemorrhages raise the periosteum, giving a typical appearance.[13]

DEFICIENCY IN RELATION TO OTHER DISEASES

The state of vitamin C unsaturation existing in pulmonary tuberculosis has been found so gross as to suggest some specific relationship. Erwin, Wright and Doherty attempted to discover the relationship, stimulated by the reports of several investigators on the improvement of tuberculous patients following vitamin C administration. These workers saturated their patients with ascorbic acid and evaluated whatever improvement might occur. After careful study they concluded that the hypovitaminosis found is a result of toxemia and non-specific. Ascorbic acid has no value in the treatment of tuberculosis or its complications. These findings are in contrast to those of Albrecht and Weber[16] who did find some improvement in allowing administration of cevitamic acid.

It has been noted that practically all cases infected with intestinal fusospirochetes have a history of a vitamin C deficiency diet. In attempting to find the correlation between these two diseases it was found that a deficiency of cevitamic acid may cause a break in the intestinal mucosa which allows for the entrance of the fusospirochete into the intestinal wall.[17]

It has been presumed that a patient on a vitamin C deficient diet would develop certain skin diseases, that is, that cevitamic acid is one of the etiologic factors. A complete and careful study was performed in order to confirm or reject this presumption by Lever and Talbott.[18] These workers determined the plasma ascorbic acid levels, and correlated their findings with the presence of a skin disease. This conclusion to this experiment was that there is no direct correlation between the level of vitamin C in the blood and the development of several diseases of the skin. Yet good results are obtained in the treatment of certain skin diseases by ascorbic acid administration.[19, 20]

Vitamin C deficiency has been incriminated as a predisposing cause to excessive lead absorption in workers who are exposed to lead. Now, however, the general consensus seems to be that deficiency of this vitamin in no way predisposes to increased lead absorption and that it is of no value when given therapeutically in increasing lead elimination.[21]

Antepartum hemorrhage in the first trimester occurs in about 9 per cent of pregnancies and is most frequently associated with the abortion state. Evidence is being accumulated showing that the antihemorrhagic vitamins C and K may be factors in the pathogenesis of certain of these cases. Vitamin C administration in these cases causes a decrease in the incidence of antepartum hemorrhage.[22]

An inadequate intake of ascorbic acid definitely has a delaying effect on wound healing. Experiments[23] have definitely proved that vitamin C is fundamental in the regeneration of tissues. The tensile strength of tissues is markedly decreased due to the imperfect formation of collagen. Vitamin C administration in deficient animals increases the tensile strength to the same degree as in normals, but in a non-defcient

animal the tensile strength is not increased.[24]

SUMMARY

The classical disease of inadequate ascorbic acid intake is scurvy, the pathology and clinical manifestations of which are well known and are described in this paper.

There is a great variation in the extent and degree of the manifestations of ascorbic acid deficiency depending on such factors as growth, stress and the degree of deficiency. Deficiency may be subclinical.

Ascorbic acid deficiency seems to have little if any relationship to other pathologic states in which it has been incriminated with exception of antepartum hemorrhage and faulty wound healing.

REFERENCES

1. Vedder, E. B.: Scurvy, Tice Practice of Medicine, 9:161, 1937.

2. Vogel, Karl: Scurvy "the plague of the sea and the opoyle of mariners," Bull. New York Academy Med., 9:459, 1933.

3. Cecil, R. L.: Textbook of Medicine, W. B. Saunders Co., Philadelphia, 1943, pp. 557-563.

4. Farrell, E.: Smollett, Lind, and Anson in 1739; their common contribution to control of scurvy, Am. J. Surg., 46:406, 1939.

5. Croft, J. D., and Snorf, L. D.: Cevitamic acid deficiency. Frequency in a group of 100 unselected patients, Am. J. Med. Sci., 198: 403, 1939.

6. Baumann, W. E., and Brock, J. F.: Vitamin C deficiency in Cape Peninsula school children, So. African J. Med., Sci., 7:212, 1942.

7. Harris, L. J.: Vitamin C levels of school children and students in war time, Lancet, 1:642, 1942.

8. Francis, G. E., and Wormall, A.: Vitamin C scurvy of medical students, Lancet, 1:647, 1942.

9. Prunty, F. T. G., and Voss, C. C. N.: Vitamin C nutrition in a hospital, Lancet, 1:180, 1944.

10. Dalldorf, G.: The pathology of vitamin C deficiency, J. A. M. A., 111:1376, 1938.

11. Moore, R. A.: The pathology of deficiency states, Med. Clin. No. Am., 27:509, 1943.

12. Mouriquand, G., Dauvergne, M., and Edel, Y.: Osteopathy from deficiency: "irreversible" decalcification of the neck of the femur in chronic vitamin C deficiency, Presse Med., 48:268, 1940.

13. Butler, A. M.: Vitamin C deficiency, Med. Clin. No. Am., 27:441, 1943.

14. Hollis, B. H.: Symposium on vitamin C deficiency, Kentucky M. J., 41:264, 1943.

15. Idem, V. P.: An internist's view of vitamin C deficiency, Idem: 270.

16. Edwin, G. S., Wright, R., and Doherty, C. J.: Hypovitaminosis C and pulmonary tuberculosis, Brit. Med. J., 1:688, 1940.

17. Woolsey, F. M., and Black, J. R.: Vitamin C deficiency and intestinal fusospirochetosis, Arch. Path., 28:503, 1939.

18. Lever, W. F., and Talbott, J. H.: Roll of vitamin C in various cutaneous diseases, Arch. Dermat. & Syph. 41:657, 1940.

19. Wolfe, M. M.: Rhinophyma with new etcologic and therapeutic considerations, Laryngoscope, 53:172, 1943.

20. Way, S. C.: Colloid milium, a vitamin deficiency, Arch. Dermat. & Syphilol., 45: 1148, 1942.

21. Evans, E. E., Norwood, W. D., Kehoe, R. A., and Machle, W.: The effects of ascorbic acid and relation to lead absorption, J. A. M. A., 121:501, 1943.

22. Javert, C. T., and Stander, H. J.: Plasma vitamin C and prothrombin concentration in pregnancy and in threatened, spontaneous and habitual abortion, Surg. Gynec. & Obstet., 76:115, 1943.

23. Beume, G. H.: Vitamin C and repair of injured tissues, Lancet, 2:661, 1942.

24. Hartzell, J. B., and Stone, W. E.: The relationship of the concentration of ascorbic acid of the blood to the tensile strength of wounds in animals, Surg. Gynec. & Obstet., 75:1, 1942.

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PATHOGENESIS OF HYPERTENSION

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INTRODUCTION

Since Richard Bright[7] in 1836 observed the relationship between albuminous urine and ventricular hypertrophy, there has been a tendency to overemphasize the connection between hypertension and renal disease. This tendency has received added impetus of late through the experimental work of Goldblatt,[28] Page,[45] Corcoran[14] and others.

It is not the purpose of this paper to attempt in any way to belittle this relationship. The purpose is rather to emphasize the fact that all cases of high blood pressure are not necessarily of an essential or a renal nature. While, admittedly, the majority of hypertensive patients will fall into these two categories, there are a number of other important conditions which will give rise to an increase in blood pressure. These should be sought for before the diagnosis of "essential" hypertension is made. It must be borne in mind that, quite often, an increase in blood pressure is a symptom of disease rather than a disease entity within itself.

Normally there are five physiologic factors operating in the human body as blood pressure determinants:[4] (1) pumping action of the heart; (2) viscosity of the blood; (3) elasticity of the arterial walls; (4) quantity of blood in the vascular tree and (5) the peripheral resistance. Uncompensated alteration of any of these factors may

lower or raise the blood pressure. In an attempt to simplify the explanation of the pathogenesis of the hypertension in various clinical disorders, a physiologic classification is presented. The disease entity associated with hypertension is classified under the hemodynamal factor whose alteration is chiefly responsible for the rise in pressure. As in any classification, there is some overlapping of categories, due to the fact that in many of the pathologic conditions more than one of the pressure determinants is affected. Clinical and pathologic classifications of hypertension have been presented by Volhard and Fahr,[61] Keith,[36] Schroeder and Steele.[56] Kahler[35] has given us one with a physiologic basis.

CLASSIFICATION OF HYPERTENSION

I. Hypertension due to an alteration in the pumping action of the heart.
   1. Thyroid disease.
   2. Tachycardia due to exertion, emotion, and so on.
   3. Aortic insufficiency.
   4. Arteriovenous aneurysm.
   5. Heart block.

II. Hypertension resulting from an increase in blood viscosity.
   1. Polycythemia vera.

III. Hypertension resulting from a decrease in elasticity of the arterial walls.
   1. Arteriosclerosis.
      a. Senile.
      b. Diabetic.
      c. Lead poisoning.

IV. Hypertension resulting from an increased quantity of blood.
   A. Generalized increase.
      1. Intravenous plasma, whole blood, and acacia.
      2. Adrenocortical tumor.
      3. Cushing's syndrome.
   B. Localized.
      1. Coarctation of the aorta.

V. Hypertension due to an increase in peripheral resistance.
   A. Associated with ischemia of vital organs.
      1. Brain.
         a. Brain tumors.
         b. Contusion of the brain.
         c. Central arteriosclerosis.
      2. Kidneys.
         a. Acute and chronic glomerulonephritis.
         b. Polycystic kidney.
         c. Hydronephrosis.
         d. Prostatic obstruction.
         e. Ureteral and urethral obstruction.
         f. Renal artery embolism or thrombosis.
         g. Mercury poisoning.
         h. Kidney infarcts.
         i. Hypernephroma.
         j. Perinephritis.
         k. Hematoma.
         l. Amyloidosis.
         m. Periarteritis nodosa.
         n. Nephroptosis.
         o. Experimental.
         p. Pyelonephritic contracted kidney.
   B. Associated with endocrine disturbances.
      1. Arrhenoblastoma.
      2. Menopausal syndrome.
      3. Pheochromocytoma.
   C. Associated with nervous disturbances.
      1. Diencephalic syndrome.
      2. "Nervous" hypertension.
      3. Bulbar poliomyelitis.
   D. Associated with an increase in body mass.
      1. Obesity.
      2. Large tumors (fibroids).
      3. Pregnancy.
      4. Myxedema.
   E. Idiopathic.
      1. Benign.
      2. Malignant.

DISCUSSION

I. *Hypertension Due to Alteration in the Pumping Action of the Heart*: Hypertension due to alteration of this physiologic mechanism is characterized by three clinical findings: (1) increase in systolic pressure; (2) low or normal diastolic pressure, and (3) an obvious increase in pulse pressure.

The predominant cause of increased blood pressure here is the increase in cardiac output, being in turn due to an increase in the minute volume of the heart. The minute volume is determined by two variable factors, that is: (1) the cardiac rate and (2) the amount of venous return to the heart. An increase in cardiac rate will not result in an increase in the minute volume of the heart without a concomitant adequate ventricular filling[4] which presupposes a greater venous return.

Thyrotoxicosis,[19] simple tachycardia due to emotion, or excitement, and arteriovenous aneurysms[26] all result in an increase in venous return to the heart. Thyrotoxicosis and tachycardia exert their effect by speeding up the circulatory rate in the body. An arteriovenous aneurysm increases the cardiac return by mechanical means. An increase in venous return causes an increase in the pressure of the great veins leading to the heart, and, through a reflex mechanism (Bainbridge reflex), the heart is accelerated in rate. Thus, both factors upon which the minute volume of the heart is dependent are increased. Since there is no increase in peripheral resistances, the diastolic pressure remains normal; or, as in the case of thyrotoxicosis, becomes lowered due to peripheral vasodilatation.

Aortic regurgitation and heart block may cause an increase in systolic pressure through a somewhat different mechanism. In these two conditions there is no increase in pulse rate. The increase in minute volume observed is due only to the increased ventricular filling. In aortic regurgitation the incompetent aortic valves allow the reflux of blood from the aorta back into the left ventricle during diastole; the heart muscle fibers are stretched, react more forcibly (Starling's Law of the Heart), and systolic ejection is more complete.[4] Thus an increased diastolic ventricular volume due to a two-way filling (mitral and aortic) plus an increase in efficiency of cardiac muscle results in an increased minute volume and an increase in systolic pressure. The diastolic pressure in this condition is obviously lowered.

Systolic hypertension associated with heart block has a very similar pathogenesis, the chief difference lies in the fact that the increased diastolic ventricular volume is due to the missing of a systolic period rather than to valvular incompetency.

II. *Hypertension Due to an Increase in the Viscosity of the Blood*: Rarely does this factor play a prominent role in the production of hypertension. Probably the only clinical condition in which the hypertension could be traced to an increased viscosity of the blood is polycythemia vera, the so-called "Geisböck" form. Many claim that in the great majority of cases exhibiting hypertension and polycythemia, the hypertension is primary and that the increased red cell count is due to a mild cardiac decompensation. Tinney, Hall and Griffin,[60] however, have fairly well demonstrated that true polycythemia vera may increase the blood pressure. They found that 40 per cent of 163 proved cases with no demonstrable decompensation had an associated hypertension.

With an increase in number of red blood cells there is an increase in viscosity of the blood and an increase in frictional resistance to flow. In the presence of an increased resistance, a constant driving force, and a constant size of the arterioles, there must result a decrease in outflow through the arterioles. Hemodynamical laws demand that, under such conditions, the pressure within the arterial tree proximal to the arterioles must rise in order to force enough blood through the arterioles to equalize the arteriolar inflow and outflow.[4]

This is the same mechanism whereby hypertension is produced when peripheral resistance is increased. The only difference lies in the fact that in the latter, arteriolar outflow is diminished by arteriolar constriction rather than by an increased frictional resistance. Similarly, also, the diastolic pressure is affected to a greater extent than is the systolic.

III. *Hypertension Due to a Decrease in Elasticity of the Arterial Walls*: Under this heading there will be no attempt made to discuss the moot relationship between

sclerosis of the small arterioles (arteriosclerosis) and hypertension. Reference will be made here only to the larger, more elastic arteries, such as the aorta and its immediate branches.

It is a well known fact that, during the normal process of senescence, these vessels undergo atheromatous and calciferous changes; suffer a loss of elastic tissue; and thus lose a large part of their resiliency. As this process progresses, the large arteries concomitantly become less able to "give" with each systolic ejection of blood from the left ventricle. The systolic blood pressure, therefore, rises. In a similar manner these inelastic vessels lose the power of "clamping down" on the smaller diastolic volume of blood. The diastolic pressure falls, remains constant, or rises only slightly. Patients with systolic-diastolic ratios in the nature of 200/90 with little evidence of renal damage or cardiac enlargement, and with peripheral and fundal evidence of extreme arteriosclerosis, should exemplify this type of hypertension.

Cerebral arteriosclerosis may produce an increase in blood pressure through the effect of cerebral ischemia on the vasomotor regulatory centers.

IV. *Hypertension Due to an Increase in Blood Volume*: Blood volume may be increased locally or generally. In either condition there will be an increase in both the systolic and diastolic pressures with a fairly normal pulse pressure.

The localized increase in blood volume is exemplified in the condition of coarctation of the aorta.[5, 55] Here, the mechanical overfilling of the arteries of the upper extremities, head, and neck give rise to an increase in blood pressure in these areas with a decrease in pressure distal to the constriction.

Steele and Cohn[51] report one case of coarctation with a generalized hypertension and suggest that, in some of these cases, the hypertension may be due to renal ischemia. They compare the hypertension in their case to that seen experimentally in constriction of the renal arteries.[28]

A generalized increase in circulatory volume is seen primarily with infusions[4] of hypertonic or protein-containing solutions, and clinically in pituitary and adrenal disorders. Some clinicians have obtained good results in the treatment of patients with essential hypertension by various methods aimed to decrease the blood volume. Pendergrass[47] noted generally a decrease in blood pressure in hypertensive patients treated by irradiation of the pituitary gland. He attributes this phenomenon to the inhibition of the pituitary antidiuretic factor, thereby stimulating diuresis and a decreased blood volume.

Hypertension associated with an adrenocortical hyperplasia[29] is generally believed to be due to the disturbance in salt and water metabolism with a concomitant increase in blood volume. Since it now seems that Cushing's syndrome is more frequently due to adrenocortical hyperplasia than to a pituitary basophilic adenoma,[29] herein may lie the explanation for the high blood pressure characteristic of this condition.

V. *Hypertension Due to an Increase in Peripheral Resistance*: Whereas the pathogenesis of an increased blood pressure noted in the preceding conditions and categories is simply explained and easily understood, it is within the present classification that much of the confusion and mystery concerning hypertension has its origin.

A great deal of experimental work has been done on the mode of production of an increased peripheral resistance, especially in renal diseases and the so-called "essential" hypertension. A survey of the literature in this respect leaves one confused in a maze of theories based upon only a few actual facts at the present time. Suffice it to say that early in all conditions associated with an increased peripheral resistance there is an arteriolar constriction which may be nervous or humoral in origin. In the presence of a constant driving force and an increased peripheral resistance, the pressure in the arterial system must arise in order to maintain the necessary relationship between arteriolar inflow and outflow. Clinically this category is characterized by an increased systolic and diastolic pressure with a variable pulse pressure depending

upon the relative degree of change of the two preceding factors.

INCREASED PERIPHERAL RESISTANCE ASSOCIATED WITH ISCHEMIA OF VITAL ORGANS

1. *Brain*: In any condition in which there is an increase in the intracranial pressure, such as in brain tumors[2] or cerebral contusion,[13] certain physiologic mechanisms come into play in order to preserve an adequate cerebral arterial supply. Practically speaking, the cranial cavity contains three fluids; that is, cerebrospinal fluid, venous blood and arterial blood. If for any reason the pressure within the cranium is increased, there is a compensatory decrease in venous blood and cerebrospinal fluid volume. The arterial supply to the brain does not decrease, however, for the pressure within the arterial tree is greater than that within the other two systems. There is, then, obviously an increase in cerebral congestion, since the arterial supply remains constant and the venous outflow decreases. A further increase in intracranial pressure thus takes place. When the intracranial pressure rises above the arterial pressure, a cerebral anemia results, with a concomitant embarrassment to the vital regulatory centers of the brain. In order for life to exist, the arterial pressure must rise. This rise is brought about through a stimulation of the medullary vasomotor centers with a resulting widespread vasoconstriction.

Cerebral arteriosclerosis is another condition that may produce a decrease in blood supply to the medullary vasomotor centers with a relative anoxemia followed by a generalized vasoconstriction. The importance of this factor is debatable, however.

2. *Kidney*: For the purpose of description, kidney conditions associated with an increase in blood pressure may be divided into circulatory and excretory disorders. Acute and chronic glomerulonephritis,[65] polycystic kidneys,[52] renal artery thrombosis and embolism,[51] kidney infarct,[1, 6, 15] hypernephroma,[48] perinephritis,[45] amyloidosis,[38] periarteritis nodosa,[11] nephroptosis,[40] pyelonephritis,[62] and chronic mercury poisoning[26] may be listed as circulatory. Conditions associated with an obstruction to the excretory products of the kidney may be prostatic, ureteral or urethral in location.[10] It has been experimentally shown (Hinman and Morrison) that the basic fault in both circulatory and excretory lesions is one and the same, renal ischemia.

It is now generally believed that renal ischemia produces hypertension through a pressor mechanism. There are three prevailing theories at the present time, and it seems justified briefly to review each of these.

In this country the theory of Page and Corcoran[1, 14, 31, 37, 42, 43, 44, 45, 46] has the greatest number of followers at the present time. As the result of a series of experiments over the past 10 years, they have concluded that the primary hemodynamic fault in renal ischemia lies in a decrease in pulse pressure in the kidney arterioles with a resultant relative arteriolar anoxia and an increase in permeability of the renal vascular tree. In the normal kidney there is a protein substance termed "renin" whose molecule is so large that the normal kidney vessels are impermeable to it. With the increase in permeability resulting from the anoxia of the vessels, this substance diffuses into the blood stream. For renin to be active as a vasoconstrictor, it must be acted upon by a substance present in the pseudoglobulin portion of normal plasma, "renin activator." The combination of renin and renin activator forms a new substance termed "angiotonin," which is the active vasoconstrictor. It is a heat stable, dialysable material. Normal plasma contains a variable amount of inhibitor to this reaction, called "renin-inhibitor." In the presence of a sufficient quantity of the latter substance, there will be no enzymic reaction between renin and its activator.

Another inhibitory group of enzymes are the "angiotonases." These are antagonistic substances, found chiefly in the kidney and liver, which react to combat the action of any angiotonin that may be formed.

Thus, it may be seen that the final rise in blood pressure is dependent upon a complicated series of "activator" and "inhibitor" type of enzymic reactions.

A group of South American workers under the leadership of Braun-Menendez[7] have arrived at similar experimental conclusions. The greatest difference between their theory and the theory of Page and Corcoran lies in terminology.

Holz,[33] in Germany, has postulated that the normal kidney contains two chief enzymes which react in the metabolism of amino-acids, decarboxylase and amino-oxidase, the latter acting only in the presence of oxygen. In conditions associated with renal ischemia, the amino-oxidase fails to act adequately and there is an incomplete deaminization of amino-acids with a resultant formation of a number of pressor substances, phenolic compounds being the most powerful of this group.

All of these are attractive theories, but at the present time remain within that realm. Suffice it to say, in conclusion, that the actual mechanism by which renal hypertension is produced remains unknown. From recent experimental work it appears to be humoral in nature.

HYPERTENSION DUE TO ENDOCRINE DISORDERS

Hypertension has been noted to appear with relative frequency in the presence of such tumors as arrhenoblastoma[21] and pheochromocytoma[22, 23] and at the menopause.[26] The means by which an arrhenoblastoma produces an increase in blood pressure is unknown; for, it is not certain as to the type of hormone liberated by this tumor.[21] It is believed to be androgenic in nature. A polycythemia has been described accompanying many of these tumors (Bingel) and could possibly be the hypertensive factor.

Pheochromocytomas increase the blood pressure by the liberation of adrenalin. The hypertension produced by this tumor is characteristically paroxysmal and may be relieved by operative removal.[9]

Although it has long been a noted fact that the blood pressure of females tends to rise during the menopause,[26] so little is known about the actual hormonal changes at this period and the effect of these changes upon the vascular tree, that, at the present, it can only be said that the hypertension is probably endocrine in origin. It may, at times, be alleviated by the administration of estrogens. Its origin is, probably, closely related to the widespread sympathetic imbalance characteristic of this period of the change in life.

Adrenal cortical tumors were not included under this heading because, although their effect is primarily endocrine in nature, the actual mechanism by which the hypertension is produced is an increase in blood rather than any change in peripheral resistance. Cushing's syndrome was omitted here for a similar reason.

HYPERTENSION ASSOCIATED WITH NERVOUS DISTURBANCES

The diencephalic syndrome of Page,[43] "nervous" hypertension,[54] and hypertension associated with poliomyelitis of the brain stem[26] probably all have their origin in central stimulation of the vasomotor centers. The diencephalic syndrome includes a group of patients who characteristically show paroxysmal attacks of blushing, cold extremities, lacrymation, hyperhidrosis, dermatographia, and hypertension. It is a syndrome similar to that produced by experimental diencephalic stimulation. The hypertension is primarily sympathetic in nature as in cases exhibiting so-called "nervous" hypertension. Individuals falling into this latter category are characterized by a blood-pressure that varies with the emotional status of the patient at any one time. It is believed that this type of person is afflicted with a hyperirritable nervous system which reacts more violently under emotional changes than does the normal person. Under ordinary conditions of living, these individuals pressure tends to vary to a much greater extent than does the average person's It is probably a precursor stage to the development of "essential" hypertension.[54] The response to emotional stimuli is probably sympathetic and adrenergic in nature.

HYPERTENSION ASSOCIATED WITH AN INCREASE IN BODY MASS

This heading was included because a number of important conditions associated with hypertension remain unclassified.

Their only common attribute is the above; namely, an increase in body mass.

The relationship between obesity and hypertension has long been noted.[39, 50, 53, 60] It has been noted also, that, in rather large series of cases, the hypertension in obese individuals is often proportional to the degree of obesity.[39, 41, 60] A reduction in body weight may result in a fall in blood pressure. At times, the degree of fall is in proportion to the amount of weight lost. It seems justifiable to conclude that, in certain cases, hypertension may be precipitated by obesity.

Exactly how this is brought about is not known. One is tempted to offer the teleologic explanation that, an increase in body mass requires some degree of increase in pressure in order that the excess tissue may receive an adequate amount of blood, since there is no increase in blood volume in these cases. It is possible that obese individuals with an inherited hypersensitive nervous system react to this new demand by an excessive increase in pressure. If this early, oftentimes transient, hypertension is long continued, it tends irrevocably to perpetuate itself. Loss of weight will then have little, if any, effect upon the pressure.

Although the relationship between large uterine fibroids and hypertension was observed in the German literature years ago with sufficient frequency to establish the term "myoma" heart,[26] very little has been written on this subject within recent years. That there is a relationship between these two conditions is well recognized.[66, 16 17, 34] There have been reports of immediate and sustained reductions in pressure following the removal of fibroids. The manner in which they act to produce hypertension in certain individuals is unknown. Everett[23] believes that renal ischemia, due to urinary obstruction from extrinsic pressure of the fibroids, explains the pressure change.

The last conditions characterized by an increase in body mass and hypertension are the toxemias of pregnancy. The cause is again unknown, although it is probably endocrine in nature. For a toxemia to develop there must be fluid retention and hypertension[19] much as in glomerulonephritis. The increase in blood pressure is not on a renal basis, however, since the renal blood flow is normal.[12, 14] As in other conditions associated with hypertension, the increased pressure tends to perpetuate itself if present for a great enough length of time. It has been shown that the development of permanent hypertensive vascular disease following toxemias is dependent upon the duration of the toxemia rather than upon its severity.[57]

IDIOPATHIC OR ESSENTIAL HYPERTENSION

All remaining cases of hypertension which do not fall into one of the previous classifications may be termed idiopathic or essential, and subclassed as "benign" or "malignant" depending upon the severity of the course and the resultant renal pathology. As has been emphasized before, any hypertension that is of sufficient duration from any cause whatsoever, or associated with any precipitating factor such as obesity, large tumors, pregnancy, and others, may become essential or self-perpetuating and remain after the eradication of the initiating factor.

SUMMARY

1. Emphasis has been laid on the fact that there are many causes for the production of hypertension and each of these conditions should be considered before the diagnosis of "essential" hypertension is made. Many may be corrected and the hypertension relieved.

2. A physiologic and clinical classification of hypertension was presented.

3. An attempt has been made to explain briefly the pathogenesis of hypertension in these various disease entities.

REFERENCES

1. Abell, R. J., and Page, I. N.: The effects of renin on vessels of ears or rabbits, J. Exper. Med., 75:673, 1942.

2. Bell, E. T., and Pedersen, A. H.: The causes of hypertension, J. A. M. A., 101:420, 1938.

3. Bell, E. T.: Renal lesions in toxemia of pregnancy, Am. J. Path., 8:1, 1932.

4. Best, C. H., and Taylor, N. B.: The Physiological Basis of Medical Practice, Baltimore, Williams and Wilkins Co., 1939.

5. Blue, J. A.: Hypertension in young athletes due to coarctation of the aorta, J. Oklahoma M. A., 36:143, 1943.

6. Boyd, C. H., and Lewis, L. C.: Nephrectomy for arterial hypertension, J. Urol., 39:627, 1938.

7. Braun-Menendez, E., and Fasciolo, J. C.: Mecanisme de l'action hypertensive du sang veineux du rein en ischeme incomplete aigni, Compt. rend. Soc. de Biol., 133: 728, 1940.

8. Bright, R.: Cases and observations of renal disease accompanied with the secretion of albuminous urine, Guy's Hosp. Rep., 1:380, 1836.

9. Brunschwig, A., Humphreys, E., and Roome, N.: The relief of paroxysmal hypertension by the excision of a pheochromocytoma, Surg., 4:361, 1938.

10. Campbell, E. W.: The significance of hypertension in prostatitis with chronic urinary retention, J. Urol., 45:70, 1941.

11. Case records of Massachusetts General Hospital No. 30201, New England J. M., 230:614, 1944.

12. Chesley, G. C., Connell, E. J., Chesley, E. R., Katz, J. D., and Glissen, C. S.: The diodrast clearance and renal blood flow in toxemia of pregnancy, J. Clin. Invest., 19:219, 1940.

13. Christopher, F.: Textbook of Surgery, Philadelphia, W. B. Saunders, 1943.

14. Corcoran, A. C., and Page, I. H.: The effects of angiotonin on renal blood flow and glomerular filtration, Am. J. Physiol., 130:335, 1940.

15. Corcoran, A. C., and Page, I. H.: Effects of renal extract containing "angiotonin" on the renal blood flow and function in normal and hypertensive dogs and humans, Am. J. Physiol., 133:248, 1941.

16. Cotte, J.: Hypertension et fibromes uterine, Lyon's med., 132:117, 1923.

17. Crossen, H. S., and Crossen, R. J.: Diseases of Women, St. Louis, C. V. Mosby Co., 1935.

18. de Takats, G., Hayes, H. E., and Keeton, R. W.: The surgical approach to hypertension, J. A. M. A., 118: 501, 1942.

19. Thompson, W. O., Dickie, L. F. N., Morris, A. E., and Hilkevitch, B. H.: The high incidence of hypertension in toxic goiter and in myxedema, Endocrin., 15:265, 1931.

20. Duff, J. L., and Murray, E. G. D.: Obstructive lesions in the main renal artery in relation to hypertension, Am. J. Med. Sci., 207:394, 1944.

21. Elden, C. A., Sinclair, F. D., Jr., and Rogers, W. C.: The Effects of toxemia of pregnancy on renal function, J. Clinic. Invest., 15:317, 1936.

22. Engel, F. L., Mencher, W. H., and Engel, G. L.: Epinephrine shock as a manifestation of a pheochromocytoma of the adrenal medulla, Am. J. Med. Sci., 204:649, 1942.

23. Everett, H. S., and Scott, R. B.: The possible etiological role of gynecological lesions in the production of hypertension, A. J. Obstet. & Gynec., 44:1010, 1942.

24. Fahr, T.: Die pathologisch anatomischen Veränderungen des Niece and Leber bei der Eklampsie, in Hinselmann, H.: Die eklampsie, Bon, Friederich Cohen, 1924.

25. Fahr, G., and Davis, J.: The effects of generalized arteriosclerosis upon the heart and systemic circulation, Ann. Int. Med., 4:211, 1930.

26. Fishberg, A. M.: Hypertension and Nephritis, Philadelphia Lea & Febiger, 1939.

27. Geist, S. H.: Ovarian Tumors, New York, Paul B. Hoeber, 1942.

28. Goldblatt, H.: Experimental hypertension induced by renal ischemia, Harvey Lectures, 33:237, 1937-38.

29. Goldzieher, M. A., and Salmovitz, S.: Endocrine aspects of hypertension, J. Clin. Endocrin., 3:37, 1943.

30. Heinbecker, P.: A role for surgery in the problem of essential hypertension, Ann. Surg., 112:1101, 1940.

31. Helmer, O. M., and Page, I: H: Purification and some properties of renin, J. Biol. Chem., 127:757, 1939.

32. Hines, E. A., Jr., and Brown, B. E.: Cold pressor test for measuring reactability of the blood pressure: Data concerning 571 normal and hypertensive subjects, Am. Heart J., 11:1, 1936.

33. Holtz, P., Credner, K., and Walter, H.: Uber die Spezifitat der Amnosaure-decarboxylasen, Ztschr. f. physiol. chem., 262:119, 1939.

34. Johnson, W. O.: A study of 104 cases of uterine fibroids associated with arterial hypertension, Am. J. Obstet. & Gynec., 43:231, 1942.

35. Kohler, H.: Die Blut druksteignerung ihre entstehung und ito mechnaismior, Ergebn. A. inn. Med. u. Kindech., 25:265, 1924.

36. Keith, M. M.: Classification of hypertension and clinical differences of the malignant type, Am. Heart J., 2:597, 1927.

37. Kohlstaedt, K. G., Helmer, O. M., and Page, I. H.: Activation of renin by blood colloids, Proc. Soc. Exper. Biol. & Med., 39:214, 1938.

38. Mark, M. F., and Mosenthal, H. O.: Kidney function and anemia in renal amyloid disease, Am. J. Med. Sci., 196:529, 1938.

39. Master, A. M., and Oppenheimer, E. T.: A study of obesity, J. A. M. A., 92:1652, 1929.

40. McCann, W. S., and Ramonosky, M., Jr.: Effect of ptosis of the kidney on blood pressure, renal blood flow and glomerular filtration, J. A. Am. Phys., 55:240, 1940.

41. Musser, J. H., and Wright, D. O.: Hypertension, obesity and hyperglycemia, J. A. M. A., 101:421, 1940.

42. Page, I. H.: Studies on the mechanism of arterial hypertension, J. A. M. A., 120:751, 1942.

43. Page, I. H.: Diencephalic syndrome, Arch. Int. Med., 190:9, 1935.

44. Page, I. H.: The effect of renal extract in lowering blood pressure in cases of essential hypertension and nephritis, J. Clin. Invest., 13:909, 1934.

45. Page, I. H.: Newer aspects of experimental hypertension in blood, heart and circulation, Am. Assoc., Adv. Sci., 13:239, 1940.

46. Page, I. H., and Helmer, O. M.: A crystallin pressor substance, angiotonin, resulting from the reaction between renin and renin activation, Proc. Central Soc. Clin. Sec., 12:17, 1939.

47. Pendergrass, E. P., Hodes, P., Jr., and Griffith, J. A., Jr.: Irradiation of the pituitary gland in posterior lobe hyperfunction controlled by biological tests, Am. J. Roentgenol., 46:675, 1941.

48. Pincoffs, M. C., and Bradley, J. E.: The association of adenocarcinoma of the kidney (Wilm's tumor) with arterial hypertension, J. A. Am. Phys., 52:320, 1937.

49. Powers, J. H., and Murray, M. F.: Juvenile hypertension associated with unilateral lesions of the upper urinary tract, J. A. M. A., 118:600, 1942.

50. Preble, W. E.: Obesity: 1000 cases, Boston M. & S. J., 188:617, 1923.

51. Prinzmetal, M., Hiatt, N., and Nageman, Y. J.: Hypertension in a patient with bilateral renal infarction, J. A. M. A., 118:600, 1942.

52. Rathburn, N. P.: Polycystic disease, J. Am. A. Genito-urinary Surg., 35:127, 1943.

53. Reily, Y. A.: Obesity and hypertension, South. Med. J., 22:157, 1929.

54. Schroeder, H. A., and Steele, A. M.: Studies in essential hypertension: I. Classification, Arch. Int. Med., 64:927, 1939.

55. Schroeder, H. A.: Studies in essential hypertension: IV. Early arterial hypertension, Am. J. Med. Sci., 204:62, 1942.

56. Schroeder, H. A.: Essential hypertension: A concept of its mechanism, Am. J. Med. Sci., 204:734, 1942.

57. Teel, A. M., and Reid, D. E.: Eclampsia and its sequelae: A clinical follow-up of all cases at the Boston Lying-in Hospital over a 20 year period, Am. J. Obstet. & Gynec., 34:12, 1937.

58. Tenney, B., Jr., and Parke, F., Jr.: The placenta in toxemia of pregnancy, Am. J. Obstet. & Gynec., 39:1000, 1940.

59. Terry, A. H.: Obesity and hypertension, J. A. M. A., 81:1283, 1923.

60. Tinney, W. S., Hall, B. E. and Griffin, H. E.: Cardiac disease and hypertension in polycythemia vera, Proc. Staff Meet. Mayo Clin., 18:46, 1943.

61. Volhard, F., and Fahr, K. T.: Die Brightsch Nierenkrankheitis klinik, Pathologic und Atlas, Berlin Jr. Spinger, 1914.

62. Weiss, E.: Failure of nephrectomy to influence hypertension in unilateral kidney disease, J. A. M. A., 123:277, 1943.

63. Weiss, S.: The development of the clinical concept of arterial hypertension, New England J. Med., 202:891, 1930.

64. Weiss, S., Parker, F., and Robb, G. P.: A correlation of the hemodynamics, function and histological structure of the kidney in malignant arterial hypertension with malignant nephrosclerosis, Arch. Int. Med., 6:1599, 1933.

65. Wosika, P. H., Jung, F. T., and Maker, C. C.: Urological hypertension as an entity, Am. Heart J., 24:483, 1942.

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SENATE BILL 637

The Honorable Allen Joseph Ellender, known to all our readers as Senator from Louisiana, has introduced a bill in the Senate of the United States which we believe should receive the endorsement of the medical profession. This bill has already been endorsed in principle by the Council on Medical Education and Hospitals of the American Medical Association and the Association of American Medical Colleges.

The bill has to do with "the release of persons from active military service, and the deferment of persons from military service, in order to aid in making possible the educational training of physicians and dentists to meet essential needs." This briefly is the purpose of the bill which provides for the deferment from the draft of a limited number of men who may be permitted to continue their medical education. Some of these men will come from the armed forces, others will be young men who have not yet been drafted but who have indicated their intention of studying medicine or dentistry. In so far as the medical students are concerned, the number shall not exceed 8000 who will be permitted to pursue first year premedical educational training. This number is reduced for second year training to 4500 and thereafter there shall not be a number exceeding the 4500 in any one of the second, third or fourth years of the medical school. The Senator from Louisiana in this bill does not limit the number of students who may be taken into medical schools who are veterans of the armed forces, women and persons not qualified for military service. The bill is planned to provide a sufficient number of doctors of medicine and dentistry necessary to meet essential needs of civilian population, especially in rural areas, and for the armed forces in the future.

This bill seems to be well considered and broad minded legislation. There is definitely an inadequate supply of physicians at the present time who are engaged in civilian practice and more particularly in the rural regions. As pointed out in a previous editorial, the need for doctors in the armed forces undoubtedly will continue for a long period of time. In the meantime accretions of civilian practitioners are very limited. The members of the medical profession now in active practice are year by year getting older and older, consequently either dying off or being forced to reduce their activities. In the states of Arkansas and Maine, for example, 25 per cent of all practitioners have passed the age of 65. Furthermore, there is a remarkable increase in the number of physicians who

wish to become specialists but a specialist cannot go into rural practice and make a living, he must confine his activities to the larger centers of population. It should be furthermore stressed that recent graduates are tending more and more to locate in the larger towns and cities of the country. Of the graduates of the class of 1938 only 18 per cent remained in rural areas.

If the needs of the armed forces even after the war will continue to be immeasurably larger than they have in the past, if so many of the young graduates wish to engage in specialties, if the men re-entering practice do not move to rural areas, and if a continuous supply of physicians is not to be turned out each year by the medical schools, it is obvious that rural areas are not going to have an adequate supply of doctors. Senator Ellender's bill is an attempt to overcome the difficulties that will have to be met to give medical care to the people of the United States if the continuous supply of doctors is not maintained. As a matter of fact if all these "if" statements made above are valid the proponents of state medicine will have excellent arguments to maintain their position because rural areas will not have doctors to take care of the illnesses and injuries of the inhabitants of these more sparsely settled districts. If for no other reason the bill of Senator Ellender should receive the support of our profession, the great majority of whom are opposed to state medicine.

A PENICILLIN PROJECT

The health departments of the State of Louisiana and City of New Orleans, with the endorsement and cooperation of the Orleans Parish Medical Society, are putting on a mass demonstration of the treatment of gonorrhea with penicillin. Penicillin is being provided by the two health departments in order to demonstrate first the value of penicillin in the treatment of gonorrhea, and secondly in order to reach those people who, because of pride, fear or for economic reasons, are being self-treated or being treated under the guidance of nonqualified individuals.

This demonstration can hardly be called an experiment in the treatment of gonorrhea because it has been definitely shown that three doses of penicillin intramuscularly given two hours apart are capable of curing gonorrhea in 95 per cent of cases. Two hundred thousand units of the drug are given in divided doses in a six hour period.

The New Orleans physicians who are participating in this study will make the diagnosis and will decide whether or not the patient has been cured. The clinical or epidemiologic diagnosis will be considered entirely satisfactory in judging the results. It will be interesting to observe and to learn of the results of this form of treatment when employed on a large scale in a city the size of New Orleans. The physicians of the city are to be congratulated on their willingness to participate in this study and to encourage an investigation, the result of which may turn out to be of tremendous importance in the control of a disease which, while not lethal, is productive of a tremendous amount of suffering and unhappiness, particularly in women.

THE SURGICAL TREATMENT OF HYPERTENSION

Undoubtedly many physicians have been quizzed regarding the surgical treatment of hypertension by their hypertensive patients since the appearance of an article in the *Saturday Evening Post* regarding sympathectomy in the treatment of this disease. Unfortunately the medical man does not have a whole lot to offer to the patient who has hypertension. The patient is told to rest, to take things quietly and possibly to take a tablet of phenobarbital two or three times a day. Some of the patients may do reasonably well on this regimen but there is a tremendous number of them who do not, as indicated by mortality statistics which disclose the fact that a large part of our population who die each year is now dying from the cardiac, vascular or renal results of continuous high blood pressure.

Smithwick,* the surgeon from the Massachusetts General Hospital who was written up by the *Saturday Evening Post*, has recently published a scientific article which discusses the result of a ten year clinical investigation of hypertension in man which has been going on at the Massachusetts General Hospital. This study has to do largely with the role of the autonomic nervous system and vascular disease as mediators of hypertension. He writes that continued diastolic hypertension is usually seen in combination with hyperactivity of the vascular bed, the latter preceding the former. There is no detailed knowledge available of the transition between the prehypertensive state to the stage of persistent diastolic hypertension. As hypertension develops there is an increase of pulse pressure with great variations in size of the change. As hypertension improves pulse pressure becomes smaller. It is the people who have what he speaks of as narrow pulse pressure type of hypertension who respond best to surgical removal of a part of the autonomic nervous system. In these individuals lumbodorsal splanchnicectomy appears to be of value. Whether this operation is going to prove ultimately to give a certain amount of symptomatic relief over a period of years will depend very largely on the selection of patients. Certainly it should not be reserved until the patient has marked eyeground changes, marked renal changes and in fact is just about *in extremis*.

*Smithwick, R. H.: Some experiences with the surgical treatment of hypertension in man, Trans. & Studies Coll. Phys. Philadelphia, 12:93, 1944.

CANCER CONTROL MONTH

April is the month designated by Act of Congress and proclaimed by the President as "Cancer Control Month." The American Cancer Society and other organizations are putting on special programs by radio and are publicizing generally the importance of cancer as a cause of death in the United States. It should be pointed out that in Louisiana it is the second ranking cause of death, as it is in the United States. In the whole country there are 165,000 people who die from this dreaded disease. It is to be hoped that members of the State Society will back up the efforts of those physicians and lay people who are publicizing the necessity of early diagnosis in the prevention of this dread and malignant disease. In so far as we know at the present time, cancer is curable only when detected early, so that its early discovery will depend upon such factors as yearly physical examinations of people of cancer age, the prompt attention to bleeding from any of the body orifices, the occurrence of indigestion in a person previously free from such symptoms, the finding of a mass in the breast, and many other indications of cancer which have not metastasized. With surgery and with radiotherapy the disease can be cured if discovered early.

ORGANIZATION SECTION

The Executive Committee dedicates this page to the members of the Louisiana State Medical Society, feeling that a proper discussion of salient issues will contribute to the understanding and fortification of our Society.

An informed profession should be a wise one.

VALUABLE MEDICAL TOPICS

We wish to make a report of the results of the various medical surveys which we have sent out to the membership in the state in order to obtain information which will be of help to physicians returning from military service. We were very gratified with the results obtained on the postcards, from which was obtained information as to the needs of different parishes and cities for medical men. This will be of value to the doctors, when they are discharged, as a guide to suitable locations and needs for same in the state.

We have not yet received replies from

forty-six parishes on the questionnaire sent to them on request of the Bureau of Information, Chicago. We urgently ask that you complete the data as rapidly as possible and return to our office, as it is necessary for us to rearrange, make additions, and supply other information before returning same to headquarters in Chicago. All of this material gathered by these surveys will be reviewed by our Committee on Postwar Planning with the object of recommending adequate post-graduate medical education and assisting in helping the physicians to re-establish themselves in practice upon their return. In order to complete this work we must have your cooperation in getting these data into our office. Most of the answers which we have received from the men in service seem to indicate a distinct desire to return to their old locations, probably needing a few weeks or a month of post-graduate work. They are trusting also to find everything just as it was before they entered the service.

There is at present a tremendous fight being waged on the medical facilities of the United States Veterans' Bureau. The public is interested in this subject and I would like to refer you to the March issue of the Cosmopolitan where there is a very interesting article entitled "Third-rate Medicine for First-rate Men" by Albert Q. Maisel. This is a very interesting discourse on the inadequate medical facilities of the Veterans' Hospitals with special reference to tuberculosis, mental diseases, and overcrowding of the institutions. Congress is also investigating veterans' facilities. The fourth interim report of the Subcommittee on Wartime Health and Education is devoted entirely to the health needs of veterans. Senator Claude Pepper, Chairman of this subcommittee, makes the following statements: "Every possible step must be taken to make certain that good medical care is within the reach of every veteran," the report maintains, and although the committee says that "its first consideration is the care of those disabled as a result of military service," its proposals indicate great concern over medical care for all veterans regardless of whether or not their disabilities are service connected.

"Criticisms of the quality of service given by the Veterans' Administration have been made to the subcommittee by organizations and individuals who speak authoritatively. It has been stated that the medical care of veterans does not measure up to the best standards.

"So important to the Nation is the assurance of good medical care for veterans that the subcommittee proposes to ascertain the facts about the quality of medical care in the Veterans' Administration. It will make a study of the present medical care program and of plans to meet the heavy responsibility with which the Veterans' Administration will be faced in the future. This study will be made at once with the help of outstanding authorities in various medical specialties, veterans' organizations, and professional groups."

Anyone can obtain a copy of this report by writing to his senator or representative. Special emphasis is being placed on the veterans' investigation due to the fact that a great many of the casualties are being returned to the United States and need hospital and medical care through the Veterans' Bureau. Such service the bureau seems to be entirely unable to supply. Many, many more hearings are expected before Senator Pepper and his committee may be ready to recommend legislation. It is believed that this committee is really attempting to obtain the best possible medical opinion before making final recommendations.

You should watch the developments of Senate bill 191 known as the Hill-Burton bill, authorizing a national survey of health facilities and providing for the construction of hospitals. This would indicate that it may be possible to arrange for adequate medical care by appropriate legislation with state levels without depending upon the supervision of the United States Public Health Service or other agencies. Hearings on this bill began on February 26, at which time the medical profession

was represented by a member of the Board of Trustees of the American Medical Association and by the Secretary of the Council on Medical Education and Hospitals. Anyone interested in reading the testimony and editorials on this important bill should see pages 652 and 656 of the Journal of the American Medical Association, March 17, 1945. It is very interesting to note that this bill is endorsed in principle by the American Medical Association and is sponsored by the American Hospital Association. The general opinion is that this is the first real scientific approach by means of national legislation toward the problem of distribution of medical care *where the need can be shown.* The significant point in this bill is that the control is left in the hands of local communities.

Recent reports would indicate that Senator Wagner is proposing to revamp his former Senate bill 1161 which was introduced in the 78th Congress. These changes would indicate that he anticipates meeting the chief objections that had been raised to the old Wagner bill. The changes call for: First, the selection of the physician and the maintenance of the relationship between physician and patient; second, it will be regulated by an Advisory Council instead of by the Surgeon General of the United States Public Health Service; third, it will call for lower tax rate. He evidently anticipates having this introduced very shortly, which will require serious study by the medical profession as to his implications and whether it would be antagonistic to the American way of practicing medicine.

Senator Ellender of Louisiana has introduced Senate bill 637, which provides for the deferment of premedical and predental students. The bill was drafted after consultation with the Council on Medical Education and Hospitals of the American Medical Association and the Executive Council of Association of American Medical Colleges. (See Journal of American Medical Association, March 10, pages 592 and 599.) This attempts to correct the current drastic regulations which resulted in a restriction in the number of students qualified to enter courses of medical instruction in approved medical schools. This bill, if passed, will relieve our apprehensions concerning the providing of adequate medical men for future civilian use.

In regard to voluntary prepayment medical insurance, which is of interest to our medical societies, you should know that recently four more states have provided for a prepayment voluntary medical insurance plan, namely, Iowa, Ohio, Connecticut, and Indiana.

The American College of Radiology has called our attention to the critical shortage of radiographic film similar to the shortage which occurred two years ago. This is due to the increased demands of the Army and Navy. They ask that every economy be practiced and that conservation of supplies now on hand be made until production catches up with demand. Hospital staffs should limit and forego the ordering of routine roentgenologic examinations, requesting x-ray studies only for cases where clinical findings warrant such procedure. It is very timely that this request be complied with in order that adequate supplies of radiographic film may be had for those who urgently need same.

TRANSACTIONS OF ORLEANS PARISH MEDICAL SOCIETY

CALENDAR OF MEETINGS

April 2 Board of Directors, Orleans Parish Medical Society, 8 p. m.

April 3 Eye, Ear, Nose and Throat Staff, 8 p. m.

April 4 Mercy Hospital Staff, 8 p. m.

April 5 Executive Committee, Baptist Hospital, 8 p. m.

April 9 Scientific Meeting, Orleans Parish Medical Society, 8 p. m.

April 11 Woman's Auxiliary, Orleans Parish Medical Society, Orleans Club, 3 p. m.

Clinico-pathologic Conference, Marine Hospital, 7:30 p. m.
Touro Infirmary Staff, 8 p. m.
April 12 Clinico-pathologic Conference, Touro Infirmary, 12 noon.
April 16 Hotel Dieu Staff, 8 p. m.
April 17 Charity Hospital Medical Staff, 8 p. m.
April 18 Charity Hospital Surgical Staff, 8 p. m.
April 20 I. C. R. R. Hospital Staff, 12:30 p. m.
April 24 Baptist Hospital Staff, 8 p. m.
April 25 Clinico-pathologic Conference, Marine Hospital, 7:30 p. m.
Catholic Physicians' Guild, 8 p. m.
French Hospital Staff, 8 p. m.
April 26 Clinico-pathologic Conference, Touro Infirmary, 12 noon.
DePaul Sanitarium Staff, 8 p. m.
April 27 L. S. U. Faculty, 8 p. m.
New Orleans Hospital Dispensary for Women and Children Staff, 8 p. m.

HOSPITAL NEWS

During the month of March the Society held one scientific meeting. The program was as follows: Comparison of Incidence and Treatment of Meningitis Over a Ten-Year Period by Drs. John H. Musser and Harry E. Rollings; A Simple Treatment for Sebaceous Cyst, by Dr. Joseph A. Danna.

NEWS ITEMS

Dr. John S. LaDue, Dr. Louis Levy, II., and Dr. Reynold Patzer attended the regional meeting of the American Federation for Clinical Research in Dallas, February 2-3. Dr. LaDue read a paper on "Possible Explanation of the Mechanism of Compensation of the Failing Human Heart;" Dr. Levy read a paper concerning "Results in the Treatment of Subacute Bacterial Endocarditis Employing Combined Penicillin and Heparin Therapy;" and Dr. Patzer read a paper, prepared by himself, Dr. Vincent Derbes and Dr. Hugo Engelhardt, on "Periarterial Infiltration in the Diagnosis and Treatment of Migraine—Experimental and Clinical Experiences with Eucupine and Procaine."

Dr. Guy A. Caldwell attended a meeting of the Advisory Board of Medical Specialists in Chicago, February 10-11.

Dr. Lewis E. Jarrett spoke on Socialized Trends at a meeting of the New Orleans Dietetic Association held February 6 at Charity Hospital.

At a recent meeting of the L. S. U. Faculty Club, Dr. R. E. Arnell spoke on Eclampsia—A Six-Year Study.

Dr. Alton Ochsner recently spoke before various medical societies in Merida, Mexico, and in cities in Guatemala, Costa Rica and Panama.

At a recent meeting of the Tulane University History of Medicine Society, Dr. Hiram W. Kostmayer was presented with a valuable pigskin-bound reproduction of Vesalius' Anatomy of the Human Body. Dr. B. Bernard Weinstein made the presentation. Dr. Alton Ochsner was the guest speaker for the society's meeting; his subject being, "The History of Thoracic Surgery."

Dr. Sam Nelken spoke on "The Social Development of Children and Children in War" at a recent meeting of the Isidore Newman Parent-Teachers' Association.

Dr. R. C. Voss was recently advanced to the position of associate medical director of the Pan-American Life Insurance Company of New Orleans.

Dr. Edgar Hull and Dr. Louis Monte recently attended the regional meeting of the American College of Physicians in Memphis. Dr. Hull, who is College Governor for Louisiana, presented a paper titled, "Bacterial Endocarditis."

At a recent meeting of the New Orleans Society of Neurology and Psychiatry, Dr. Theo A. Watters was elected president; Dr. Sam Nelken was elected secretary.

At the January monthly meeting of the Medical Staff of DePaul Sanitarium the following officers were installed for the year 1945: Dr. Edmund Connely, president; Dr. T. A. Watters, vice-president, and Dr. Louis J. Dubos, secretary-treasurer.

Major Hardee Bethea and Captain John S. Herring, both stationed in the Pacific area were home on leave recently.

Lt. Richard Corales, Jr., is now stationed at the U. S. Naval Hospital in New Orleans.

Lt. Jack C. McCurdy received the Navy Cross "for extraordinary heroism in action" against the enemy on Guam, July 21, 1944, while serving as battalion surgeon with the United States Marines. Rear Admiral A. C. Bennett, U. S. N., commandant of the Eighth Naval District, made the presentation at ceremonies held February 8 at the Houma Naval Air Station. Before entering the service, Lt. McCurdy was a resident at Baptist Hospital.

It has been reported that Major C. Barrett Kennedy is ill and confined to a hospital in Italy.

DE PAUL SANITARIUM STAFF MEETING

The following report was received from Dr. Louis J. Dubos, Secretary:

The Regular Monthly Meeting of the Medical

Staff of De Paul Sanitarium was preceded by the annual dinner, and called to order at 8:55 p. m., with Drs. Fuchs, Cole, Thompson, H. Colomb, A. Colomb, May, Otis, Holbrook, Anderson, Watters, Blum, Graffagnino, Friedrichs, Golden, O'Hara, Connely and Dubos in attendance.

There was no presentation of cases at this meeting, so Dr. Otis summarized in detail the reports of the various departments for the entire year of 1944, all of which showed satisfactory progress.

When requested by the Chairman to say a few words, Sister Anne expressed her sincere appreciation of the work of the members of the Staff during the past year and their hearty co-operation in meeting and maintaining the approval of the American College of Surgeons and thereby placing De Paul Sanitarium on the "approved list" of hospitals where it justly belongs.

In reviewing the progress of promoting psychiatric nursing, Sister Anne said that the program of 1944 had not been as successful as the program of 1943 and had failed to achieve its goal —that of establishing a School of Psychiatric Nursing. Such a school would of necessity have to be set up on the same level as the other Schools of Nursing in New Orleans—that is, on a university level, particularly if the students of Charity Hospital were to come into it, since they have a university affiliation. Sister Henrietta concurred in this opinion, suggesting that Charity Hospital nurses be sent up to De Paul Sanitarium for practical training, management, teaching and field practice if such a permanent course could be arranged.

A motion was made by Dr. Friedrichs and seconded by Dr. Holbrook that a committee be appointed to write a letter of appeal to the Mother Provincial of the Sisters of Charity in St. Louis, requesting her assistance in establishing such a school by sending a Sister to De Paul Sanitarium for the definite purpose of organizing a nursing school program.

Dr. Golden stated that he hoped that residencies in neuropsychiatry would eventually be established at De Paul Sanitarium as well as the other general hospitals in the city because up to the present time no specialized neuropsychiatrist has ever been trained here in New Orleans.

Sister Anne informed the entire Staff that she had acquired the services of an excellent occupational therapist whose services would be available daily.

There being no further business, the meeting was adjourned at 9:50 p. m., followed by a meeting of the New Orleans Society of Neurology and Psychiatry.

LOUISIANA STATE MEDICAL SOCIETY NEWS

CALENDAR

PARISH AND DISTRICT MEDICAL SOCIETY MEETINGS

Society	Date	Place
East Baton Rouge	Second Wednesday of every month	Baton Rouge
Morehouse	Second Tuesday of every month	Bastrop
Orleans	Second Monday of every month	New Orleans
Ouachita	First Thursday of every month	Monroe
Rapides	First Monday of every month	Alexandria
Sabine	First Wednesday of every month	
Second District	Third Thursday of every month	
Shreveport	First Tuesday of every month	Shreveport
Vernon	First Thursday of every month	

EAST AND WEST FELICIANA BI-PARISH MEDICAL SOCIETY

After an excellent dinner in the East Louisiana State Hospital dining room, the society repaired to the Staff Room for a scientific program.

Electric shocks were given to several patients, and presentation of cases on constitutional psychopathic personality, paranoid dementia precox simple type, psychoneurosis and paresis.

The program, which was enjoyed by all present, was presented by Drs. Strum, Green, Nolan and Robards.

Dr. Toups of Baton Roue, Louisiana, was elected honorary member of our society.

A vote of thanks was extended to Dr. Glenn J. Smith, and staff, for their entertainment.

Society adjourned to meet the first Wednesday of June, 1945.

B. F. Smith, President
E. M. Toler, Secretary.

RAPIDES PARISH MEDICAL SOCIETY

At a recent meeting of the Rapides Parish Medical Society the following officers were elected for 1945: President, Dr. H. H. Hardy, Jr., Alexandria; First Vice-President, Dr. F. A. Thomas, Urania; Second Vice-President, Dr. Ed-

mond Klamke, Alexandria; Secretary-Treasurer, Dr. R. U. Parrott.

ACADIA PARISH MEDICAL SOCIETY

The following officers were elected at the meeting of the Acadia Parish Medical Society held in Crowley on February 21: President, Dr. U. J. Arretteig, Church Point; Vice-President, Dr. G. G. Fontenot, Morse; Secretary-Treasurer, Dr. A. A. Williams, Church Point.

IBERIA PARISH MEDICAL SOCIETY

At a recent meeting of the Iberia Parish Medical Society the following officers for 1945 were elected: President, Dr. Henry J. Dauterive; Vice-President, Dr. Harold M. Flory; Secretary-Treasurer, Dr. P. M. Payne; Delegate, Dr. W. P. D. Tilly, all of New Iberia.

NEWS ITEMS

Dr. G. W. McCoy, Professor and Director, Department of Public Health, Louisiana State University School of Medicine, attended the National Foundation for Infantile Paralysis meeting in New York City, March 13-14, and a meeting of the U. S. Pharmacopoeial Revision Committee in Washington, D. C., March 16, 1945.

The American College of Chest Physicians has cancelled its 11th Annual Meeting, which was to have been held in Philadelphia, June 16-19.

The American Psychiatric Association, the oldest Medical Society in America, has announced the cancellation of their 101st Annual Meeting, which was to have been held in Chicago, in May of this year. It was the feeling of the Association that it would be the duty of the membership to fall in line with the request of the United States Government to cancel conventions in the spirit of the war cooperation.

The annual banquet of the Southern Baptist Hospital Staff was held at the Jung Hotel Roof, March 27, at 8 p. m.

The general oral and pathological examinations of the American Board of Obstetrics and Gynecology will be conducted in Atlantic City, June 14-19. The Hotel Shelburne will be the headquarters for the board.

ARMY MEDICAL RESEARCH BOARD

Lieutenant Colonel Roy H. Turner, M. C., Chief, Communicable Disease Treatment Branch, Medical Division, Surgeon General's Office, has been placed on the Executive Committee of the Army Medical Research Board, of which Major General George F. Lull, Deputy Surgeon General, is President. Brigadier General Stanhope Bayne-Jones, a former New Orleanian, is also on this Executive Committee. Also on the committee of eight is Lieutenant Colonel Michael E. DeBakey, Chief, General Surgery Branch, Surgical Division of the Surgeon General's Office.

POSTGRADUATE COURSE IN MEDICINE

A postgraduate course in "Recent Advances in Therapy" was held under the auspices of the Department of Graduate Medicine of the Tulane University of Louisiana School of Medicine, March 5-10. This course was under the direction of Dean Kostmayer, with Professor George Burch in charge of arrangements. The course was attended by 61 men, five from Central America, with representatives from seventeen states. Five of the attendants came from the state of Michigan, three from Illinois, one from South Dakota, as well as from practically every state south of the Mason-Dixon line.

The program was participated in by members of the faculty of the School of Medicine, with two guest speakers, Dr. Raymond Gregory, Professor of Medicine at the University of Texas School of Medicine, and Dr. Irving H. Page, Director of Research at Cleveland Clinic, Cleveland, Ohio.

HEADQUARTERS, EUROPEAN THEATRE OF OPERATIONS, U. S. ARMY

Award of the Bronze Star Medal to Capt. S. E. Morgan of New Orleans, La., has been announced by the 101st Airborne Division.

Learning of several wounded soldiers badly in need of medical care, Capt. Morgan braved heavy enemy mortar and small arms fire to reach the forward position in which they were. When enemy attacks forced other men of the position to withdraw, Capt. Morgan supervised the evacuation of less seriously injured, and elected to remain with those casualties who were too dangerously injured to be moved, despite the risk of capture or death.

ALPHA OMEGA ALPHA

The annual banquet preceding the induction of new members into the Stars and Bars Chapter of Alpha Omega Alpha medical honorary fraternity was held at the Hotel Jung on Monday, March 19. In addition to the old members and the initiates, the three officers of Alpha Omega Alpha chapter at Louisiana State University Medical Center were guests. Dr. Joseph Menendez acted as toastmaster and the toast of the evening was delivered by Dr. Alfred E. Cohn, Member Emeritus of the Rockefeller Institute for Medical Research.

On the following night Dr. Cohn spoke on "Disease of the Heart" at a public meeting. Succeeding this talk the members of the Senior class at Tulane elected to A. O. A. were inducted into membership. They were: Thomas G. Baffes,

Rodney C. Jung, John J. Baehr, William D. Franklin, William H. Blahd, Beverly Blood, Gerald N. Weiss, Gerald S. Berenson, Roy White, Jr., George W. Prather and Merrill S. Prows.

ARMY ACHIEVING SPEEDY EXPANSION OF HOSPITALS

The Army's expansion of its general hospitals by 70,000 beds is being rapidly accomplished through the conversion of existing buildings on hospital grounds rather than through new construction, according to the Office of The Surgeon General.

"At many of the general hospitals," said Brigadier General Raymond W. Bliss, U. S. A., Assistant Surgeon General, "there are well-constructed barracks, built with an eye to the future, which were used to house overseas hospital units during their training period. These barracks are now being turned into wards for patients. Permanent barracks, built to house the hospital staff, are also being converted into wards and are being replaced with temporary barracks which can be quickly constructed."

Over 50,000 more patients are being cared for in the Army's general hospitals than was the case three months ago. During the past month about 1,200 casualties arrived from overseas daily.

MASS CHEST SURVEYS

The American College of Radiology announces that their policy in regard to mass chest surveys is as follows:

"The American College of Radiology approves the principle of mass chest surveys for the detection of pulmonary tuberculosis for public health purposes.

"The films should be examined by physicians who are trained and competent to interpret radiographs of the chest.

"Cases showing abnormalities should be immediately referred for further study and care to local physicians, or, when this is impractical, to other available agencies.

"In case-finding surveys conducted as a public health measure by eleemosynary agencies, the fee for roentgen interpretation of survey films should be a sum approximately equal to fifteen dollars per hour, said fee to be a matter of agreement among radiologists in the area concerned."

These releases were reaffirmed at the meeting held September 27, 1944, in a conference with Dr. Herman S. Hilleboe, Director of the Division of Tuberculosis Control of the United States Public Health Service.

INFECTIOUS DISEASES IN LOUISIANA

The morbidity report of the Louisiana State Department of Health showed that for the week ending February 10 the following diseases were reported in numbers greater than 10: Measles 35, malaria 31, chickenpox 28, unclassified pneumonia 25, scarlet fever 18, pulmonary tuberculosis 17, mumps 15, diphtheria 11. It is rather unusual to have as many as 10 cases of diphtheria reported. This week six of these cases came from Vermilion Parish. Most of the cases of malaria were reported from military sources; as a matter of fact, 28 were contracted outside the Continental United States. For the week which ended February 17, chickenpox was first in the number of cases listed by the State Board of Health. There were 65 cases followed by 48 of malaria, 31 of whooping cough, 28 of unclassified pneumonia, 26 of pulmonary tuberculosis, 24 of measles, 17 of scarlet fever, and 16 of dysentery. The diphtheria cases were scattered over the whole state. Of the malaria cases, 46 were contracted outside of the Continental United States. For the following week, February 24, malaria again was very high with 32 cases, and 28 of them were from military sources. Of the purely local diseases there were 35 cases of tuberculosis, 25 of chickenpox, 21 of unclassified pneumonia, 20 of scarlet fever, 19 of measles, and 18 of mumps. No unusual or rare diseases have been reported during the month of February. The morbidity report for the week ending March 3 contains the monthly statistics of the venereal diseases. In that period of time there were 1,420 cases of gonorrhea listed, 1,145 of syphilis, granuloma inguinale 31, 21 of chancroid, and 12 of lymphopathia venereum. There are two remarkable features of this report aside from the large number of venereal disease cases that were reported, namely, that 151 cases of cancer are listed, and 153 of unclassified pneumonia. This was decidedly a pneumonia as well as a venereal disease report because there were listed also 48 cases of pneumococcic pneumonia. All these pneumonia cases came from the civilian population except one. Other diseases reported in numbers greater than 10 include 67 cases of pulmonary tuberculosis, 52 of chickenpox, malaria 41, rheumatic fever 34, septic sore throat 17, scarlet fever 16, measles 15 and mumps 14.

HEALTH IN NEW ORLEANS

The Bureau of the Census, Department of Commerce, reported that during the week ending February 10 there were 165 deaths in the City of New Orleans as contrasted with 152 of the previous week. Ninety-eight of the deaths were in the white and 67 in the colored population, with nine of the deaths occurring in children under one. The following week the figures were very close to those of the previous week. Of the 159 deaths taking place in the city, 107 were white, 52 non-white, with 11 deaths in small children. For the week closing February 24, 151 people expired in the City of New Orleans. The number of white deaths dropped to 86 and the

non-white increased to 62. Thirteen of those who died were small children, mostly colored. For the week which ended March 3 there were 141 deaths, divided 90 white, 50 non-white, with a very considerable number of the total deaths being children. This time the infant mortality was high among the white children with 12 of them expiring and only five in the colored population. For the week which closed on March 10 there was a decided improvement in the number of deaths, only 113 being reported, separated by races, 70 white, 43 colored. There were 11 children under one year of age dying this week.

DR. RALPH HOPKINS
(1876-1945)

One of the outstanding medical men of New Orleans passed away on the seventh of March. Dr. Ralph Hopkins was known not only as a splendid dermatologist but he undoubtedly was the outstanding authority on leprosy in this country. For forty-three years he had been associated with the United States National Leprosarium at Carville. It was said of Dr. Hopkins that except when he served in France in the last war that in this period of time he had not missed more than three or four weekly visits to the institution.

Dr. Hopkins was professor of dermatology at Tulane for many years. He was vice-president at the time of his death of the American Dermatological Association.

A kindly man, skilled in his profession, he was universally liked and respected. It was said of Hopkins that he had no enemies.

DR. HENRY AUSTIN MACHECA
(1896-1945)

The many friends of Dr. Henry A. Macheca heard with sorrow of his death Monday, March 19, following a heart attack. On account of ill health Dr. Macheca retired from active practice five years ago. Prior to that time he served on the staffs of Touro and Charity Hospital, and had taught at the two medical schools in New Orleans. He has been a member of the Orleans Parish and the State Medical Societies for many years, as well as being a member of the American College of Surgeons and the Phi Chi Medical fraternity.

WOMAN'S AUXILIARY

REPORT OF THE WOMAN'S AUXILIARY TO THE LOUISIANA STATE MEDICAL ASSOCIATION FOR MARCH 1945

News from the Woman's Auxiliary this month brings activity in scope both national and local. Busy Mrs. Rhodes Spedale, as president, went to Chicago to attend a conference of State Presidents, Presidents-Elect, and Chairmen of Standing Committees of the Women's Auxiliary to the American Medical Association. The mid-year meeting of the State Auxiliary Board was held at the home of the president in Plaquemine, and a majority of the parish groups have been visited by her.

The purpose of the national conference at this time was to promote the exchange of ideas of mutual interest and help to state auxiliaries. Although state programs must follow the national outline in general, plans and problems which arise in local groups are of cumulative value when discussed with others.

Mrs. John P. Helmick, whose charm and personality will be remembered from her visit at the time of the auxiliary convention last spring, presented greetings to the group from the Woman's Auxiliary to the Southern Medical Association. Mrs. David W. Thomas, president of the Woman's Auxiliary to the American Medical Association, directed the activities of the conference. Mrs. Jesse D. Hamer, of Arizona, was introduced to the group as president-elect of the national auxiliary.

The following were elected as officers of the meetings: Mrs. Roscoe Meisman, Washington, Conference Chairman; Mrs. Lee J. Schaefer, Kansas, Secretary; and Mrs. Fidler, Ohio, Chairman of the Committee on Recommendations.

Contact of the state group with the Woman's Auxiliary to the Southern Medical Association was furthered through a visit of Mrs. W. W. Potter, president-elect of the latter organization, to New Orleans, as guest of Mrs. Paul Lacroix, where she was also met by Mrs. Spedale.

Shreveport, Ouachita, Rapides, East Baton Rouge, and Lafourche Auxiliaries have been on the travel itinerary of the Louisiana president. Vermilion and Acadia parishes have been reorganized! They are enthusiastically welcomed into the parent organization.

All parishes are further urged to "disseminate information and educational material regarding the Wagner, Murray-Dingell bill" and copies of said bill have been mailed to every state officer, chairmen of standing committees, and parish presidents.

April has been designated as Cancer Control Month, and every doctor's wife is expected to take an active part in the campaign. Mrs. H. A. Thompson, State Commander of the Women's Field Army, has cited several auxiliary members for the valuable time they have given to this worthy cause. A still higher goal for alleviation of needless suffering and death is the aim of the coming drive.

The celebration of the Fourth Anniversary of the U. S. O. was made complete with auxiliary representation through Mrs. Paul Lacroix, president-elect.

In addition to the many phases of war participation, doctors' wives should work unitedly as never before in promoting health education, public relations, in fighting tuberculosis, and in commemoration of Doctors' Day on March 30. Editors of the New Orleans newspapers have promised cooperation in the latter activity with cartoons and editorials on that date, and parish chairmen are in a position to suggest similar publicity with ideas concerning present projects of their groups.

Upon recommendation of Mrs. Spedale, the president elect of the association will hereinafter be also the chairman of organization. This chairmanship will enable her to have closer contact with the auxiliary members before taking office. She will not only be in a position to organize new auxiliaries, but to contact members of disbanded groups, and to encourage the active societies to a more progressive status.

It is with extreme regret that notice must be given of the plans for a Louisiana State Medical Meeting this year. It was at first thought that a business meeting of the auxiliary might be permitted at the same time as the one day session of the House of Delegates of the doctors' group, but it is now definite that neither will be possible this year.

It is therefore at the special request of Mrs. Spedale that minutes of the Board meeting are being printed as recorded so that those absent will know exactly what took place. A careful perusal of the agenda is urged of all members. Parish presidents and chairmen of publicity are cordially invited to send material of interest to the group to the state chairman for inclusion in these monthly reports.

Respectfully submitted,
(Mrs. Edwin R.) Mazie Adkins Guidry,
Chairman of Press and Publicity.

EXECUTIVE BOARD MEETING

February 6, 1945.

A meeting of the Executive Board of the Woman's Auxiliary to the Louisiana State Medical Society was held on February 6, 1945, at the home of the President, Mrs. Rhodes J. Spedale, in Plaquemine, La.

Mrs. Spedale called the meeting to order and the assembly rose to unite in saying the Lord's Prayer.

The secretary called the roll of officers, chairmen of standing committees, and parish presidents. The following members were present:

Mesdames Rhodes Spedale, Paul LaCroix, Arthur Long, Carroll Gelbke, R. D. Martinez, Frank Jones, E. C. Melton, Kelly Stone, M. C. Wiginton, John S. Dunn, C. Grenes Cole, Daniel Murphy, J. E. Heard, Roy B. Harrison, Roy C. Young, Theodore Simon, Wiley A. Dial, Aynaud Hebert and Lloyd Kuhn.

Mrs. Spedale introduced Mrs. Paul LaCroix, President-Elect, and announced that Mrs. LaCroix had represented the State Auxiliary at the celebration of the U.S.O. Fourth Anniversary, and that she had also entertained Mrs. W. W. Potter, President-Elect of the Southern Medical Auxiliary.

The minutes of the Post-Convention Board meeting were read. Mrs. Murphy asked that the phrase "Study of the Wagner-Murray-Dingell Bill" be changed to "Opposition to the Wagner-Murray-Dingell Bill," in the outline for the program for 1945. With this correction the minutes stood approved.

Mrs. R. D. Martinez, Treasurer, presented the treasurer's report with a balance of $385.22. This report was filed with the consent of the assembly.

Mrs. Arthur Long, Vice-President, took the chair while the President gave her report. Mrs. Spedale reviewed the work done by the auxiliary to date in 1944-45. She urged all Parish Auxiliaries to remain organized even if it is necessary to be inactive due to decreased membership. Eighteen parish auxiliaries were reported as active this year. Mrs. Spedale reported that she attended the A.M.A. Convention in Chicago, and that Mrs. Cole, Mrs. Harrison, Mrs. Peacock, Mrs. Donovan Browne and Mrs. Herald attended also. Mrs. Herald was made Constitutional Secretary at that time. Mrs. Spedale and Mrs. Herald also attended the Fall Conference. In conclusion Mrs. Spedale made the following recommendation:

> "that the President-Elect be made Chairman of Organization. This chairmanship will enable her to have closer contact with Auxiliary members before taking office. In order to reap any benefit from said chairmanship she must be active, not only in organizing new auxiliaries, but in contacting auxiliaries that have disbanded and encouraging the active ones to a more progressive status."

Mrs. LaCroix moved, seconded by Mrs. Cole, that the recommendation be accepted. Motion passed unanimously. The President's report was placed on file.

Mrs. E. C. Molton, Corresponding Secretary, reported 72 cards and letters written, and 35 copies of the Wagner-Murray-Dingell Bill mailed to Parish Presidents and officers of the Auxiliary.

Councilors' Reports

Mrs. Theodore Simon, First District Councilor, reported that district fully organized and functioning satisfactory.

Mrs. Roy B. Harrison, Second District Councilor reported the reorganization of that district with nine members. The new officers were elected as follows: Mrs. John Atkinson, Gretna, Pres-

ident; Mrs. William Guillotte, Vice-President; Mrs. Earl Clayton, Recording Secretary; Mrs. P. A. Donaldson, Treasurer.

Mrs. Spedale commended the work of the Second District.

These reports were placed on file.

Reports of Chairmen of Standing Committees

Mrs. Lloyd Kuhn, Chairman Cancer Control, reported that she attended the first meeting of the state committee and announced that materials and letters will be sent to each parish auxiliary.

In the absence of Mrs. Taquino, Chairman of Doctors' Day, Mrs. Spedale reported that letters have been sent to each parish auxiliary asking for reports concerning their plans for Doctors' Day.

Mrs. Wiginton, Fiance Chairman, read the budget for the year.

Mrs. John S. Dunn, Historian, reported that letters have been sent to each parish president asking for pertinent illustrations and articles.

Mrs. Aynaud Hebert, Chairman of Indigent Widows' Fund, reported one donation from Iberville Auxiliary and three personal donations making a total of $36.00.

Mrs. C. Grenes Cole, Chairman of Legislatoin, reported on the work in opposition to legislation regarding socialized medicine.

Mrs. David Murphy, Chairman of Printing, reported that the Year Book supplements and the stationery had been printed within the budget allowed.

Mrs. Heard, Chairman of Public Relations, requested that we publicize the radio proram entitled "Doctors Look Ahead."

Mrs. Young, Chairman of Revision of By-Laws, stated that the revision of the A.M.A. Constitution affects in no way the State Constitutions.

These reports were filed by consent of the assembly.

Reports of Parish Presidents

In the absence of Mrs. Sandidge, Mrs. Heard read the report for Caddo Parish which revealed a very active and successful year for that auxiliary.

Mrs. Arthur Long, President East Baton Rouge Parish Auxiliary, read her report with emphasis on war work and civic activities for that group.

The Iberville Parish report was given by Mrs. R. J. Martinez, President, showing varied activties and reporting 14 hundred dollar War Bonds purchased by the auxiliary.

Mrs. John Dunn gave an oral report of the work done by the Orleans Parish Auxiliary up to January 1945.

Under new business Mrs. Young read the President's report of the first conference of State Presidents, Presidents-Elect, and Chairmen of Standing Committees of the Woman's Auxiliary to the American Medical Association, held in Chicago, November, 1944.

This report was placed on file.

The secretary read two letters from Dr. Talbot regarding the State Convention. He reported that the Executive Committee had decided to have a one day meeting of the House of Delegates on April 13, rather than the annual four day session of the Society. The Auxiliary was advised to secure permission from the War Committee on Conventions if the officers desired to have a meeting at the same time as the House of Delegates. The Secretary then read a letter from Mr. R. H. Clare, Secretary Committee on Conventions stating that it is present policy of the Committee to deny application form.

Mrs. Hebert moved that we make tentative plans for a one day convention at the same time as the meeting of the House of Delegates. Mrs. Long seconded the motion. It passed unanimously.

It was the pleasure of the Board not to submit an application to the War Committee on Conventions as in all probability the attendance will not exceed fifty persons.

As there was no further business, Mrs. Stone moved, Mrs. Long seconded, that the meeting stand adjourned.

After the meeting the members were guests at a delightful luncheon in the home of Mrs. Rhodes Spedale.

Respectfully submitted,
Mrs. Frank J. Jones, Recording Secretary,
Woman's Auxiliary to the Louisiana
State Medical Society.

BOOK REVIEWS

Handbook of the Mosquitoes of North America: By Robert Matheson, Ithaca, N. Y., Comstock Publishing Co., Inc., 1944. Pp viii, 314, illus. Price, $4.00.

The present war has brought to the fore the importance of malaria and other arthropod-borne diseases as military and homefront hazards. As a result there are probably more individuals directly concerned with the control of mosquitoes in this country and the world over than at any other time in history. It is particularly fortunate, therefore, that Doctor Matheson, Professor of Entomology at Cornell University, has produced a new, revised and enlarged edition of his authoritative "Handbook of the Mosquitoes of North America" which, since its original appearance in 1929, has been a valued guide to students of this branch of entomology. While the literature on North Amer-

ican mosquitoes is formidable in bulk and rich in detail, monographic works of continental scope have been few and long outstripped by rapid advances in knowledge. Here, in this slim volume of 314 pages, is condensed the fruits of many years of patient labor, in the form of a convenient handbook that is essential equipment for mosquito-workers in the United States and Canada. It makes readily available the essence of our present knowledge of the taxonomy and biology of North American mosquitoes, much of which could otherwise be found, if at all, only in many separate publications and in older lengthy and cumbersome treatises now difficult to obtain.

The book is divided into two sections. Part I (86 pages) is a general survey of the structure and biology of mosquitoes, their relation to human welfare, their control, and methods of collecting and studying them—subjects of interest not only to the entomologist, but to epidemiologists, health officers, physicians, and laymen, who will find here a clear and readable source of information. Part II (172 pages) is a systematic account of the North American species of mosquitoes—primarily of interest to the entomologist as a working tool.

The first part includes a detailed account of the morphology of the adult and larval stages which is fundamental to practical work of identification. The life cycle, breeding, feeding and overwintering habits, knowledge of which is essential to practical control work, are briefly reviewed. The role of mosquitoes in the transmission of human malaria, yellow fever, dengue, filariasis, and virus encephalitides is concisely treated, with the malaria parasite cycle described and illustrated. The fundamental principles of control are outlined and the various types of control methods considered. Directions for collecting and preserving mosquitoes for study conclude this section of the book, which contains 41 good drawings and photographs to illustrate the textual material.

The second part, more technical in nature, provides means to identify systematically the larvae and adults of North American mosquitoes by appropriate keys to subgroups and species, and describes their group and specific characteristics. Descriptions of 133 species are given, distributed among 15 genera. Nearly half of these are species of *Aëdes* which include some of our worst day-biting pests of marshes and woods, occurring in untold numbers in the Arctic and along our coasts. *Aëdes aegypti* transmits yellow fever and dengue, and *A. cantator* is an important vector of eastern encephalomyelitis. The genus *Culex*, with 19 species, includes the common night-biting "house mosquitoes," which can transmit filariasis, and *C. tarsalis* which has been found infected with the viruses of St. Louis encephalitis and western equine encephalomyelitis. Thirteen species of malaria mosquitoes, *Anopheles*, are recognized in North America, *A. quadrimaculatus* and *A. freeborni* being the most important vectors because of abundance and feeding habits. While anophelines may be distinguished from culicines by easily detected characters, specific identification of mosquitoes requires the study of minute details of structure, color pattern, and (in larvae) of hair arrangement. Such necessarily detailed descriptions are given, with notes on the known distribution, breeding, and feeding habits of each species. Illustrations are provided in 33 plates of excellent photographs of typical breeding places and beautifully executed drawings of larvae and male genitalia. The latter afford the best characteristics for specific identification.

This edition includes over 200 classified bibliographic references as a guide to the literature on mosquitoes of America and other regions. It is interesting to note that about forty more species have been recognized in North America in the fifteen years since the first edition appeared, reflecting our increased knowledge of this important group of insects. (In 1901 L. O. Howard included only 25 species in the first general work on U. S. mosquitoes!)

The necessarily brief treatment of mosquito-borne diseases and control measures includes only passing mention of the newer developments in these fields, but due note is made of the advent of aerosols, synthetic repellents, and DDT. These promising methods of attacking mosquitoes have been largely the result of intensive war research and already bid fair to rank in importance with sulfonamides and penicillin as weapons against disease.

The book is printed on good quality paper in a neat and attractivce format, with good typography designed for ease of reading and reference, and with an adequate index.

While much of this volume is technical and specialized and will be used mainly by entomologists, everyone interested in the part mosquitoes play in public health and welfare will profit by reading the more general chapters. The book is a mine of useful and authoritative information. At this time it is more than ever evident that the practical aspects of mosquito-knowledge are literally a matter of life and death. Broadly conceived and sound academic studies such as this are fundamental to practical progress and provide part of the essential power in the complex machinery of combatting some of the more material ills that beset man.

ALBERT MILLER, Ph.D.

PUBLICATIONS RECEIVED

Charles C. Thomas, Springfield, Illinois: Neuro-Ophthalmology, by Donald J. Lyle, B. S., M. D., F. A. C. S.